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HEREDITY

BY

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THIRD EDITION

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PREFACE TO THE THIRD EDITION

An almost complete rewriting of this book has been necessitated by a combination of developments, partly of human genetics, partly of experience. The general criterion by which a genetic phenomenon was admitted to the first edition was the demonstration, probability, or belief of some responsible person that that phenomenon occurs in man or is involved in the economic features of some cultivated plant or domestic animal. While that criterion still partially guides the choice of topics, the number of proved and suspected genetic phenomena in the human race has grown so rapidly in recent years that the same policy now leads to a general elementary treatise. Practically all the simpler genetic situations must be included, and some not so simple. One combination of modes of inheritance not usually presented in elementary treatises is described because a human trait has been held to rest upon it. The original purpose of the book is expressed in the free use of examples of heredity in man to illustrate genetic principles. Practically every pertinent chapter, every discussion of a genetic phenomenon, includes one or more human cases in point. These do not replace the several chapters formerly devoted to human heredity, but are in addition to them; the chapters referred to give information regarding more characters than ever before.

Other extensive changes relate to exposition and sequence. Experience has indicated that there are improved methods of presentation. Chief among these, the author believes, is the inclusion of the chromosomes in the diagrammatic explanations of experimental results at the very outset. Just as the chemical elements are best understood by a direct approach from electrons and protons, without reference to the periodic table, so genetic results are most easily interpreted with a full knowledge of the gene and chromosome mechanism in advance. That is the order here used. The traditional method has been to present

experimental results, formulate a scheme of gene operations which will logically explain them, and then—long afterward—show that these operations call for genes which are located in the chromosomes. While this has been the order of discovery, few sciences are most effectively mastered by following the sequence of their historical development. In this edition the evidence that the genes are in the chromosomes is still given, more fully than before, but late; and in the meantime, from the very beginning, the knowledge that they are there has been used in the interest of clarity.

Certain changes have been made in the order of presentation. Sex-linkage is presented early. Of other phenomena, those having a monofactorial basis, even some formerly treated as complications, are described first, before those involving two or more loci. Biometric methods have been transferred to an appendix, where they will not appear to break the continuity of courses not making use of them, but in that position the discussion is strengthened. Instead of merely cultivating an appreciation of the need of statistical treatment in certain problems, and of the general nature of the aid they offer, this postponed account should enable the student to perform the more common operations with a minimum of guidance and with some understanding. Teachers who use statistical methods can introduce them at any convenient point.

At the request of users of the former editions, a list of literature, referred to by author and date throughout the text, has been added. To save space, chiefly the literature of recent years is included, with the idea that these publications will in turn cite the earlier work. If this practice appears to give credit where it is not due, the author can only plead in extenuation that the purpose of a bibliography is to guide the reader to the literature.

Problems and questions of more or less objective types are introduced in considerable numbers at the end, where they follow closely the order of the several chapters. While questions of these kinds do not call for organization, they may be made to require thought, and because of the brevity of their answers make possible a comprehensiveness of test not attainable through answers of the essay type. These questions are not numerous enough to cover the text; they suggest rather what teachers may prepare in addition.

It is believed that the style of the new book will be found factual, but organized. To the thoughtful consideration of teachers and students the author commits it, with the hope that it will be of service.

A. FRANKLIN SHULL.

ANN ARBOR, MICHIGAN,
March, 1938.

EXTRACT FROM PREFACE TO THE FIRST EDITION

This book has been gradually developed . . . in connection with a lecture course given to large classes of college students who are admitted to it without prerequisite. A majority of the members of this class have had no previous training in biology, and a very great majority of them seek a general education. The eager inquiry of such students has seemed to the author an opportunity for missionary work seldom surpassed in college. Their interest centers largely in human affairs. To cultivate that interest in wholesome directions, to establish their social outlook on valid foundations, and to prepare them to cope with problems, some of which they will probably live to see become acute, are tasks that challenge the wisdom and discernment of any teacher.

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HEREDITY

CHAPTER I

DEVELOPMENT OF KNOWLEDGE OF GENETICS

The development of any science has usually taken essentially the same course. Isolated observations are first made, most often at long intervals, by keen-witted folk who recognize that some rule must lie at their foundation. Others less discerning, now that their attention has been called to these phenomena, note further examples, and a body of related facts is accumulated. Eventually someone sees or imagines a scheme into which at least part of the observed facts fall, and a theory is developed. To support or refute this theory, a deliberate search for additional data is made, and the body of knowledge grows more rapidly. The added information requires new (often supplementary) theories, which grope among the facts for confirmation and many of which have to be abandoned. Stimulated by these speculations, inquirers resort to experiment, and discovery grows apace. Finally, by good luck, industry, and cunning, perhaps by the application of new discoveries in some other field, someone may lay bare a rather fundamental principle on which all or many of the facts rest, and a great advance is made.

Geometrically, this history of the science could be represented by a curve of exponential type, rising slowly at first but with gently increasing speed, then rapidly, and finally with a rush amounting almost to verticality. Some sciences have traveled this entire path, others stand now at various points along it. In biology, genetics has outdistanced most other branches and may fairly be said to have approached more nearly to the status of an exact science than have any of the other divisions. How has it attained that distinction among the biological disciplines?

Early Ideas.—The long period during which this curve of knowledge rose barely perceptibly is marked chiefly by indirect evidence that heredity was known to exist and that practical

advantage was taken of it. Sculptures and drawings show that the ancient civilizations of Babylon and Assyria, several thousand years before the Christian era, had brought horses and cattle measurably near their modern state of advancement, though it is not known whether this improvement was due to deliberate selection with betterment in view or was merely a result of choosing the best individuals each generation for their own immediate value. Only in the former case would a knowledge of heredity be proved. Not much later, however, that is, about 5000 years ago, rice was being grown by the Chinese, and their ancient writings show that some varieties were regarded as better than others. Since varietal differences could be maintained only by heredity, it seems certain that this phenomenon was then recognized. Grains were cultivated by the Egyptians at least as early as 4000 B.C., for grains of barley have been found in mummies of that time; and wheat, rice, soybeans, flax, and cotton have never, even in the remotest records, been referred to as wild plants—they have been cultivated during all that time. It would be difficult to imagine that crops could be grown in complete ignorance that heredity exists, but mere husbandry does not prove knowledge of it.

Coming down to the classical Greek period, including the fifth, fourth, and third centuries before the Christian era, we find many references to subjects related in one way or another to heredity. They have been assembled by McCartney (1927). Most of these references indicate ideas, but not knowledge, for the statements made are recognized now as wholly mistaken. For example, inheritance of acquired characters was quite regularly assumed; and characters were supposed to be acquired in the most unlikely ways. Some simple observations were more or less correctly made, as when Hippocrates averred that children with blue eyes are born of blue-eyed parents, and that bald heads and squint eyes in one generation lead to bald heads and squint eyes in the next. Certain eye diseases were seen to recur in particular families, the descendants of the Corinthian Timoleon being subject, like himself, to blindness in old age. Aristotle noted, as one may now do, that when a child does not resemble its parents, it may be like its grandparents or more remote ancestors. With at least partial truth the ancient Scots held that "falling sickness, madness, gout, leprosy"

are likely to be "propagated from the father to the son," and drastic measures were sometimes adopted to prevent those afflicted from handing them on. By far the greater number of notions of heredity belonging to these early times were, however, erroneous, even fanciful. The idea that inherited characters could be modified or even supplanted by some subtle influence of the mother's passing thoughts was prevalent. Mothers, by looking at a portrait, were believed to be able to make their children resemble neither parent. Not alone man, but horses, sheep, and pigeons were held to be subject to such impressions. In Old Testament times, this alleged principle was used by Jacob, as he believed, to swell the wages promised him for the care of Laban's flocks (Genesis 30:30–42), though, if the facts are correctly stated, they are now known to have an explanation in certain other well-established features of heredity.

Beliefs Concerning Method of Heredity.—How transmission took place, or how maternal impression or other environmental influence modified it, received only occasional attention. Hippocrates held that heredity occurred part by part—that healthy parts are derived from healthy ones, unhealthy from unhealthy. To explain this piecemeal constitution of the offspring, it was natural to assume that representatives of some sort separately derived from the organs were collected and handed on to each descendant, and this view was held not only by Hippocrates but by his contemporary Democritus. It is paralleled by Charles Darwin's theory of pangenesis of less than a century ago, according to which gemmules, collected from the organs into the germ cells, are dispersed again to corresponding organs for whose nature they are responsible. Aristotle, however, only a century later than Hippocrates, reversed the process and regarded the heredity units, not as coming from organs to the reproductive germ, but as going from this germ to the various organs not merely of the same individual but of the next generation. The latter part of this idea is in essential agreement with the concept of heredity today.

Environmental influences which modified heredity must have been supposed to act upon the physiology of development. At any rate, some such agents could conceivably so act. Thus mothers who ate too much salt in their food were said to beget children without nails. Sex was supposed by Aristotle to depend

on the "form-building power" of the respective parents; this power declined with age, so that old fathers would have mostly girl children. Aristotle also held sex to depend on temperature, and a cold north wind was thought to be female-determining.

These notions belong to the early Greek civilization. They are largely expressed again by the Romans of the first Christian century, through the prolific pen of Pliny The Elder who copied them from the Greeks—at any rate, copied them—without any significant additions. Since the dark ages that followed hard upon the Roman period yielded little in any intellectual province, we may turn back to gain a deeper insight into the backwardness of the heredity of the Greek and earlier eras.

Reproduction in Animals.—At even the remotest times, the knowledge and delusions of which have here been portrayed, there was probably no doubt in the minds of men regarding the general method of reproduction in animals. No people given to the rearing of domestic animals could have failed to recognize the existence of sex and to learn that two parents are regularly necessary to the production of offspring. If heredity was observed at all, it must early have been traced to two potential sources in each instance.

An engraving on bone in an excavation in Mesopotamia, dating from about 2800 B.C., is interpreted by Amschler (1935) as a pedigree chart of horses of several different types. Aristotle recognized that crosses between unlike individuals constitute the chief means whereby heredity could be studied. He knew that the mule is the result of a cross between the horse and the ass, and supposed, perhaps erroneously, that the hybrids from this cross differ, depending on whether the horse is the sire or the dam. He mentions likewise that the mule is sterile, and speculates upon the anatomical reasons for the sterility. In this knowledge Aristotle was not particularly forehanded, for the sterility of the mule was known to the Babylonians several thousand years earlier. It is often said that the Romans (Varro, Pliny) knew not only that the mule is sterile, but that some mules are fertile (as is now known to be true). It is easy, however, to misinterpret the brief references in the ancient works, and Zirkle (1935) inclines to the view that the fertile mules of Varro and Pliny were some other kind of animal. No increase in knowledge concerning mules was made for at

least a thousand years, for essentially the same statements about them are made in a Persian cyclopedia published in A.D. 1339 or 1340. The famous Greek philosopher suspected—for what reason is not clear, though the suspicion was correct—that the honeybee is parthenogenetic and appreciated that under these circumstances the offspring would derive from only one parent. He records also the belief in crosses between the dog and the wolf or fox, without, however, apparently knowing anything about them at first hand.

Lest these ancient philosophers be credited, from the foregoing list of achievements, with greater keenness of perception than they actually possessed, it should be added that they also "knew" many things which could not have been true. Animals possess, as they thought, very generalized powers of reproduction, so that crosses between unlike types were held possible. Thus the old Greeks implicitly believed that the tiger sometimes crossed with the dog; serpents with eels; lions with various other mammals. They were indeed a credulous people. It was only necessary that statements appear in writing to win for them full recognition, for no one ever investigated them. One might therefore be inclined to think that, if the ancient Greeks managed sometimes to tell and believe a true tale of natural history, that was good fortune; but the successes credited to them in the several preceding paragraphs appear really to have been earned, not merely accidental.

Sex in Plants.—If sex in animals was patent to everyone, that of plants was most elusive; and therein lies one of the reasons for the slowness of progress in acquiring a knowledge of heredity. For the workings of heredity are in general simpler in plants than in domestic animals, once it is learned how to discover them. Moreover, plants are so much less expensive to rear, that an inquiring naturalist might satisfy his curiosity concerning plant heredity when he could not afford to do so with animals. So long as sex was not recognized in plants, the chief simple and easy road to a knowledge of heredity was thus closed. The crossing of varieties could not well be effected or observed when the method of reproduction was unknown.

The first plant to throw light on this subject was probably the date palm, grown by the Babylonians 6000 years ago. It was known that there were two kinds of trees, one of which

FIG. 1.—Hand pollination of date palm, as portrayed in Assyrian bas-relief. (*From Zirkle, The Beginnings of Plant Hybridization, University of Pennsylvania Press.*)

bore fruit if, and only if, the other kind was present. These two kinds were of course the female and male, respectively. Canny horticulturists at least as early as the ninth century before Christ knew that pollen is the agency through which the male induces fruit bearing in the female, for works of art of that period portray the process of hand pollination (Fig. 1).

Sex in other plants was not then discovered, presumably because in so many of them, including those most commonly cultivated by man, there were both male and female parts on the same plant, usually in the same flower. There was in these species only one kind of individual, hence nothing conspicuous to attract attention to the pollination. The lesson of the date palm was not carried over to plants as a group, and it was many centuries before sex came to be recognized as of general occurrence in plants.

Aristotle, Herodotus, Pliny, and Theophrastus, covering the period from 484 B.C. to A.D. 79, all mentioned the supposed existence of sex in plants, but apparently had done nothing to confirm the belief or to show how widespread the phenomenon is. According to Sarton, St. Thomas Aquinas says something that seems to indicate a knowledge that plants have sex. That was in the thirteenth century. During the next several centuries a number of writers indicate a suspicion, if not knowledge, of the function of pollen, referred to as dust or as some more subtle influence. Many of their works are quoted by Zirkle, "The Beginnings of Plant Hybridization," and by Roberts, "Plant Hybridization before Mendel." Some biological historians credit Camerarius with the first real proof of plant sex in 1694, but that advance was made gradually; and even the work of Camerarius was either unknown or unconvincing to his contemporaries and successors, at least in other lands, for as late as 1759 the Imperial Academy of Sciences of St. Petersburg was enough in doubt about it to offer a prize for the best solution of the problem of sex in plants. That prize was won by the Swedish botanist Linnaeus with a paper entitled "Disquisitio de Sexu Plantarum."

Early Hybridization.—It would naturally be supposed that hybridization of plants would wait upon and follow knowledge of their sex. As a matter of fact, however, the two went hand in hand. Knowledge of sex came mostly through observations

of hybridization; and the real nature of hybridization was understood only as sex was revealed. With this discovery the curve of knowledge of heredity began to rise a little more rapidly. Some ancient beliefs regarding hybrids have already been described. Now that the machinery of crossing was known in such inexpensive things as plants, inquisitive and acquisitive gardeners could be depended on to produce them at will.

Such amateur plant breeding was, indeed, carried on, before the nature of pollination was understood, by gardeners who merely observed that when plants of different varieties were planted near one another they sometimes produced individuals that were intermediate or of mixed qualities. The annals of England and of the English colonies in America in the seventeenth and eighteenth centuries contain many examples of such observations, which are described, with quotations, by Zirkle. There are too many of them to mention here individually, but particular reference should be made to Thomas Fairchild who in 1719 crossed a carnation with a sweet william, since this is apparently the first hand pollination specifically on record.

These naturalists and gardeners approached the phenomenon of hybridization from several different angles. The early observers of hybrids which were produced accidentally by neighboring plants noted the mixture of the parental qualities and regarded it as a sign of degeneration. A little later to some naturalists hybridization was of interest chiefly as a proof of sex in plants. To those who were classifiers of plants, the capacity to cross bore distinctly on the question of the real distinctions between varieties. To the more worldly-minded, as well as to those aesthetically inclined, hybridization offered the means of improvement of plants to human ends. And to some staid folk the deliberate effecting of crosses seemed an unwarranted and unsavory interference with the processes of nature.

Kölreuter.—This period of development of ideas of sex and hybridization was climaxed, and a new era begun, with the work of Kölreuter (Fig. 2) in Germany. If the background for his work, consisting of the earlier observations and experiments already narrated, is understood, the advance made by Kölreuter will be appreciated. He undertook his experimenting with plant crosses with a seriousness not previously attempted.

Beginning with a cross between two species of Nicotiana (tobacco) in 1760, he followed with 65 other crosses involving many different species. Kölreuter was interested in the processes of heredity, for he described the characters of the hybrids and compared them with the characters of the parents. Sometimes the hybrids were intermediate, sometimes like one of the parents.

FIG. 2.—Joseph Gottlieb Kölreuter (1733–1806), leading early hybridist. (*Journal of Heredity*.)

Some hybrids were sterile, some showed greater vigor than the parents. Kölreuter's object was not essentially different from that of geneticists today, and his papers were prepared with much the same care as are present-day scientific articles. Indeed, he is frequently referred to as the first scientific hybridizer.

Approach to Mendelism.—The remainder of the story of genetics gathers closely around the fundamental tenets comprised in what is now known as Mendelism. Every important advance made during this period either led to the laws propounded by Gregor Mendel or was built upon them. To understand what was happening during this time, it will be

necessary to anticipate briefly the essential features of Mendel's laws, which are developed in later chapters.

Organisms must not be thought of as wholes, but as composed of many parts or characters. These characters are transmitted somewhat independently of one another, by means of heredity units. These units come to an individual from each of its parents, hence exist in pairs within that individual. But when they are in turn handed on to the next generation, the units separate (segregate is the word commonly used) so that only one of each pair goes to any one of the offspring. Since the heredity units relating to a given character need not be identical, it follows that the offspring of any given parents may differ from one another, depending on which units each has received from its two parents. Moreover, the units concerned with different characters may and commonly do segregate from their twins independently of one another. Consequently, individuals may be made up of very different combinations of characters, arrived at through processes which are largely random.

This is the scheme of heredity propounded by Mendel in 1865 and 1866. It has been proved to be in general correct, and Mendel has accordingly been revered as the prophet of modern genetics. Now, most important advances in science, invention, and the like have their forerunners. Hero-baiting historians dig up observations made earlier which resemble the great discovery to a degree, and claim with some show of plausibility that it was all done before. That has happened to Gregor Mendel, and the inference has been drawn that perhaps his honor has been excessive. Since most of his discovery is broadly related to the segregation of heredity units, we may look among the records for signs that others before him understood the situation.

Signs of Segregation.—One of the consequences of segregation, as has been pointed out, is that individuals belonging to certain hybrid generations differ from one another. This variability had been observed long before Mendel. It was seen in tulips in 1576, in peonies in 1588, and in carnations in 1633, where it was regarded as a process of degeneration. These and others like them are mentioned by Zirkle. It was seen in 1588 in corn (Fig. 3), where it is now known to be due to hybridization

though not necessarily to segregation. Bradley in 1717 stated that many different types arose among hybrid primulas, while differently colored peas in the same pod, following accidental crossing, were reported by Henchman in 1731. Other similar observations followed over the years. Linnaeus's prize paper of 1760, mentioned earlier, was concerned primarily with proof of sex in plants but incidentally described the hybrids on which that proof rested. In them the fruit might be like one parent, the leaves like the other. Kölreuter's Nicotiana hybrids, of about the same time or a little later, also broke up into different combinations.

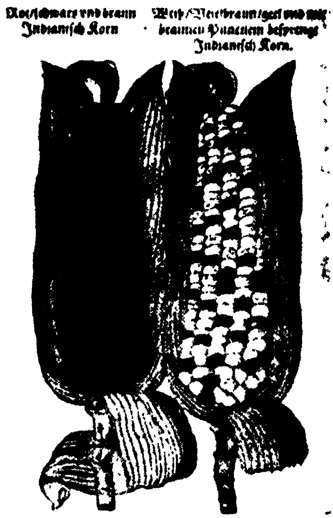


FIG. 3.—Variation among progeny illustrated in corn in 1588, to be attributed either to segregation or to mixed paternity. (*From Zirkle, The Beginnings of Plant Hybridization, University of Pennsylvania Press.*)

The first naturalist to align the characters of the two parents in definite pairs was Sageret in France, who crossed muskmelons and cantaloupes before 1826. Their characters were listed as flesh, white or yellow; skin, smooth or netted; flavor, acid or sweet. In the hybrids these qualities were combined in different ways. Some academy prizes helped stimulate interest in these questions; one was a belated offer in 1819 and again in 1822 by a Prussian academy of sciences for definite proof whether plants really do hybridize—a question satisfactorily answered long before—and another was by a Dutch academy in 1830 for a study of the possibility of creating new varieties by crossing. The latter question bore directly upon segregation, for new varieties produced by segregation would be very different from those produced without it.

Gärtner in Germany, hearing of the Dutch prize, entered the competition with studies he had already made on various plants and won it in 1837. In these studies and others, up to 1849, he found that in one respect the hybrid leaned toward one plant, in another respect toward the other. In 1845 Lecoq had observed the variability of hybrids and referred to it as "shattering the

stability of the type," obviously considering the variation to be lawless. A prize offered in 1861 by the Paris Academy of Sciences brought forth works by Godron and Naudin. The latter won the prize, and his paper was of the more interest in the present connection; for the chief use made of his study by Godron was to distinguish between species and varieties, while Naudin placed emphasis on the shuffling of the parental characters and their recombination in a variety of ways in the hybrids. Naudin observed that first generation hybrids were fairly uniform (all individuals much alike), but that later generations produced from these hybrids were often quite diverse. He described a hybrid as "an individual in which are found united two different essences . . . which are incessantly in a struggle to disengage themselves from one another." This disengagement resulted in what seemed to him "disordered variation." Laxton in England, in experiments with peas which began in the early 1860's, observed many different combinations of flower colors, seed coat colors, seed shapes, and stem heights; but, being interested chiefly in determining the origin of the cultivated kinds of peas, he did not regard a full analysis of the hybrids as particularly valuable. The assortment of independent characters was also noted by the Vilmorins (father and son), who were better known for their practical breeding of sugar beets and wheat, and for their adherence to the principle of pure line breeding which has since been generally adopted.

The work of Nägeli, celebrated German botanist, though mostly done at least as late as the preceding breeders, was really a recession from their positions. Nägeli was interested in the distinctions between species and varieties and regarded a species as a single entity not to be analyzed into distinct characters whose fate in hybrids must be separately followed.

Mendel's Criticisms.—Mendel was familiar with a number of the experiments just described. He had read all the accounts of such experiments which were available to him. And he was not satisfied. Writing of Gärtner's experiments, he says:

It is greatly to be regretted that this meritorious investigator failed to publish a detailed description of his particular experiments or to undertake an exhaustive diagnosis of the various forms of hybrids. Such remarks as that "some individuals resembled the maternal and others the paternal type more closely," or that "the offspring were in

general more of the type of the mother of the first generation," etc., are couched in too general terms, are too vague, to justify the grounding of a definite opinion upon them.

Similar objections could be urged against all the other experiments. What Mendel required, in order to understand the operations of heredity, was that each individual plant should be on record as having this sort of stem, that color of flower, a certain shape of seed and a given form of seed pod, and then to know that, out of a large total number of hybrids in each generation, a certain number of plants had each particular combination of characters. He pointed out that not one of the previous investigators had analyzed each hybrid, down to its separate characters, and that not one of them had given the precise number of individuals of each kind.

To remedy this situation, Mendel decided to do some intensive experimentation himself. After due consideration of the species used by others and after some preliminary trials of his own to determine what plants would best furnish the necessary information, he elected to work with garden peas.

The Famous Pea Experiments.—Garden varieties of peas already in existence furnished many different characters ready to use. Stems were tall and dwarf in different varieties; seeds were green or yellow, also round or wrinkled; pods were inflated or constricted between the seeds; seed coats were either colored or colorless; the pods green or yellow; and the flowers were either distributed along the stem or they were bunched at the tips. Plants differing in only one of these respects were crossed in the simpler experiments, but more characters were simultaneously used later. Throughout his experiments Mendel was contrasting, not one whole plant with another whole plant, but each character with a corresponding character. When two pure varieties were crossed, all their offspring were alike; and if the varieties had differed in just one character, the hybrids were usually just like one of the parent types. Thus, if yellow-seeded peas were crossed with green-seeded ones, the hybrids were all yellow-seeded. It made no difference which of the varieties had been used as the female, which as the male, for the hybrids were the same, yellow-seeded, in both instances. When, however, these yellow hybrids were self-fertilized, their offspring were of the two original kinds, some yellow, some green; and

out of very large numbers (over 8000 in this particular cross) almost exactly 3/4 were of one kind (yellow), 1/4 of the other (green). Crosses involving the other characters yielded numerically the same results—the first generation hybrids all alike and like one of the parents, the second generation of two kinds in the ratio of about 3 to 1.

When plants differing in two characters were crossed, their hybrids again were all alike and might be like one parent in both respects, or like one in one character and like the other in the other character, depending on how the two qualities were combined in the parents. When these double hybrids were self-fertilized, the next generation was of different kinds; this time there were four kinds, and again they bore a definite numerical relation to one another. What this ratio was may well be left to a later chapter, but it was essentially the same ratio in every experiment in which the original parents differed in two respects.

Mendel was thus obtaining the precise information about hybrids which he had looked for in vain in the writings of his predecessors. To explain the numerically similar results obtained with respect to all the different characters, he invented the scheme of segregation which has already been briefly outlined. He postulated heredity units, upon whose nature he wisely did not speculate, but which he was content merely to symbolize with letters, *A* and *a*. Two of these units related to any given character were present in each plant, but, before they were transmitted, they separated (segregated) so that only one went to any one offspring. In the offspring one such unit entered with the female contribution and one with the male, but which type of unit came with each one was determined at random. It was this segregation of the heredity units and their random recombination which led so regularly to the ratio of 3:1 among the offspring in the second hybrid generation or to the more complex but equally regular ratios if two or more pairs of characters were involved.

Mendel's Priority.—Enough has been told of the work of Mendel and of his predecessors to form now a judgment regarding the implied suggestion that his conclusions had been anticipated by others. Segregation of outward characters had indeed been witnessed long before Mendel's time. It had not, however, been reduced to any regular scheme. There is, moreover, a wide gap

between observing the segregation of visible characters and postulating the segregation of the responsible internal units. Naudin had, it is true, reached the conclusion that it is in the pollen and the ovules of plants where the "disengagement" of the "two different essences" contributed by the parents must take place. But he had no conception of a plan in accordance with which the separation occurred. Neither he nor any of the

FIG. 4.—Gregor Mendel, from portrait in Moravian Mortgage Bank (*From Iltis, Life of Mendel.*)

other early hybridizers possessed the sort of information which would suggest a scheme or by which any scheme could be tested. Nothing short of a complete analysis of each hybrid, character by character, and precise numbers of individuals of all kinds would suffice. Such information Mendel had, because he obtained it himself; all earlier workers lacked it. It seems clear, therefore, that the fundamental part of Mendel's scheme was new with him, and that he is entitled to the honor commonly bestowed on him as the father of modern genetics.

Story of Mendel's Life.—Descendant of a long line of gardeners, Johann Mendel (Fig. 4) was born in 1822. In school, the short, stocky lad stood at the head of his class, marked "eminent" in progress and "very good" in all the branches of study—but in the religious lectures merely "good." Despite the latter slight deficiency, he was recommended to an Augustinian monastery

at Brünn, in what was then Austria, now Czechoslovakia, in 1843, and was accepted. There he assumed the monastic appellation Gregor, by which he was thereafter known. The monastery was a center of learning, and, when the modest, not particularly reverent, Gregor proved to be temperamentally unsuited to priestly duties, he was shunted to the educational program. Though he had avoided the natural history course, in his early school work, in favor of physics, Mendel was always interested in natural phenomena and was at different times a sunspot observer, a weather bureau operator, a mouse breeder, a microscopist with flower parts the objects of study, an apiarist even to the extent of crossing different varieties of bees—all in addition to the famous work in plant hybridization. It was to botany that his chief energies were devoted in the monastery school, and, though he twice failed in an important examination in that and other subjects at the University of Vienna because he had been merely self-taught, he nevertheless managed to make valuable contributions to it in later life. His duties as teacher were not too heavy to permit the plant-breeding experiments which made him famous, and these were an important part of his work over at least a 10-year period.

Soon after the publication of his monograph on the pea experiments, events began to shape themselves in opposition to his continued scientific work. For one thing, the now rotund, short-winded professor was no longer able to scour the countryside without limit on botanizing trips. Moreover, so respected was he by all concerned that in 1868 he was chosen prelate to head the monastery. Thereafter he became more and more immersed in administrative work and gave less and less to his plant crosses. He also had an unfortunate controversy, extending over a number of years, with the state on the question of taxing monasteries. During this dispute he became somewhat estranged from some of his colleagues, who thought that yielding a principle might have relieved the institution of some of its financial difficulties. Mendel maintained his position of defiance, however, until his death in 1884. His declining years were thus rendered bitter as well as unproductive.

Neglect of Mendel's Work.—Since evolution had become a burning question through the publication of Darwin's "Origin of Species" in 1859, and since heredity is an important element of the

evolution process, it might be supposed that Mendel's pea experiments would be promptly seized upon by naturalists and used to the full. That was not their fate, however; instead they were completely ignored for a third of a century. Darwin himself never heard of Mendel's work. The only biologist who is known to have been acquainted with it at the time was the botanist Nägeli. A number of lengthy letters passed between Nägeli and Mendel concerning the pea crosses, but the former seemed not greatly impressed, and commented that the experiments, "far from being finished, are only beginning." The only references to Mendel's experiments in the three decades following were one by Hoffman in 1869 and another in a paper by Focke in 1881, in the latter of which it is stated that his work was much like that of his predecessors but that "Mendel believed he had found constant numerical ratios among the types produced by hybridization." It was not until 1900 that his paper was resurrected, after others had discovered the same principle of segregation and recombination.

Various explanations have been offered for this 34-year neglect of Mendel. The suggestion that the Proceedings of the Brünn Natural History Society was an obscure publication is scarcely valid. Iltis, in his "Life of Mendel," holds that the conciseness of Mendel's paper was against it; readers could hardly convince themselves that anything so small could really be of much value. A third view is merely that the time was not ripe, that the biological world had not yet been brought up to the point where work of the precise and mathematical-looking sort done by Mendel appeared promising. The general interest of naturalists in evolution and natural selection following the publication of Darwin's "Origin of Species" was doubtless part and parcel of this unripeness of the times for experiments in heredity. Finally, it has been suggested that Mendel might have won recognition for his idea if he had advertised it; and the reason why he did not advertise it was that he feared it was not of general application. There was some reason for such fear; for, after getting well along with the peas, Mendel had also tried hybridizing the hawkweeds, Hieracium, and these plants gave very erratic numerical results. Biologists know now that Hieracium is to some extent parthenogenetic, so that what Mendel regarded as hybrids were not really such. This would

account for the irregularity. Mendel knew nothing of this parthenogenesis, and was at a loss for an explanation. The suggestion made is that Mendel himself did not believe he had hit upon a general law (Hatch 1933) hence did not drive for its recognition. His innate modesty would have helped him reach such a decision.

The Rediscovery of Segregation and Discovery of Mendel.—Whatever the reason for the neglect, Mendel was not discovered until biologists were ready to rediscover his law independently. That state was not reached until the late years of the nineteenth century. The incentive to renewed study of heredity came from knowledge of variation. Crossing is of significance only if individuals differing in some respect are mated. Studies of variation, notably those conducted by Bateson and De Vries, revealed individual differences which could be so used. Consequently, in the 1890's the attack on the heredity problem was renewed. De Vries, of Holland, found new variations arising in the evening primrose but used also many other kinds of plants in crosses. Correns, in Germany, used corn, peas, beans, lilies, and stocks. In Austria, von Tschermak worked on peas. There were others, but these three are mentioned particularly because of the curious coincidence that they reached the publication stage almost simultaneously, that they arrived at the Mendelian law independently, and that in their search through the literature they all independently discovered Mendel's old paper.

De Vries was the first to publish, in March, 1900. He had learned of Mendel's paper from a list in Bailey's "Plant Breeding," the citation there having been taken from Focke's paper of 1881. Correns, having finished enough experiments to reach conclusions, quickly published them (May, 1900) when he saw the paper of De Vries. Only a few weeks later was the publication of von Tschermak. Correns and von Tschermak had both found Mendel's paper from the reference to it by Focke. All three of these botanists had been led to adopt the principle of segregation of heredity units and the random recombination of these units to explain their own new results. They were of course not a little surprised to find that similar results and a similar conclusion had been on record since 1866.

Modern Genetics.—The success of these plant hybridizers stimulated great interest, and biologists everywhere began to

test the new-old hypothesis in a variety of plants and animals. The law of segregation received, from these tests, fairly general support. With this additional backing, interest in it rapidly grew. Biologists in other fields viewed the flourishing parvenu "genetics" a bit skeptically, and some of them declared it could not be what it was being pictured; the scheme was too simple. And so it was. It was not long before it was found that the system of Mendel had to be modified.

One of the earliest changes was necessitated by the discovery that in some animals, though one parent transmitted the segregated heredity units to all their offspring, the other parent handed on certain units only to half their offspring, and that half were all of one sex. In some species it was the male, in others the female, that possessed this limited capacity of transmission.

Another change was required when it was found that certain characters were not sorted out with entire freedom, but that some of them tended to hang together. Correns had observed this in his work prior to 1900, and others found it later. Consequently, a mechanism whereby the heredity units could be bound together, not too unerringly, in the cells had to be found. Fortunately a mechanism, the chromosomes, which would do this was plainly at hand.

A third modification was needed when it was found that two parents visibly differing in only one respect might produce, two or more generations later, more than two kinds of descendants. A single difference was splitting up into several differences. It had to be supposed in these instances that two or more pairs of heredity units were cooperating, in the original parents, to produce one visible effect, while due to the shuffling that took place in later generations they entered into situations where each unit operated singly or at least with other groups.

Thus the scheme grew and developed. Yet through all these changes the essential Mendelian feature remained—the segregation and recombination of the units of heredity. These units have been located in the chromosomes of cells, and it has been found that they have a very specific arrangement there. Chromosomes have been broken, and the break carries with it a corresponding change in the heredity of certain characters. Chromosomes have been lost or added, and some character or other is modified to suit. So many such peculiar situations have

been found or produced and the inherited character so closely connected with a minute intracellular change, that it is now possible to look in a microscope at the cells of certain organisms and point almost exactly to the spot where something lies that is responsible for an eye color, a wing shape, or the number of joints in a leg.

Important Objects of Study.—Though most organisms whose heredity has been studied agree fundamentally with the above plan, some have yielded more particulars than others. The most fruitful of all objects of such study has been the vinegar fly Drosophila. Changes that could be used in making crosses began to crop up in 1910, and since then about a thousand such modifications have arisen. Under the leadership of T. H. Morgan, a group of investigators has pushed knowledge of the heredity of this fly far beyond anything that would have been dreamed of as possible 20 years ago. Corn has been used by a large group of cooperating and independent workers, and its genetics is only moderately less well known than that of Drosophila. The evening primrose Oenothera, De Vries's source of plant changes, has a rather specialized scheme of heredity, which has been intensively studied by a large corps of botanists on both sides of the Atlantic.

Among the higher animals, only those which are not too expensive to rear can be used for large-scale studies. These are mice, rats, guinea pigs, and rabbits. Mammals being more complicated, these studies have yielded fewer details than have plants or the simpler animals; but they have the advantage of standing nearer to man, and so perhaps of indicating more closely the trend of human problems of heredity than does Drosophila, corn, or Oenothera.

Human Heredity.—Inheritance in man has not been overlooked, but it cannot be used advantageously to discover the details of the heredity mechanism. What is known of man's heredity is therefore mostly an application of what has been discovered more plainly and earlier in other organisms. The first important student of human heredity was Sir Francis Galton, working in the last third of the nineteenth century while Mendel's paper lay unnoticed, hence without the example of complete individual analysis furnished by the pea experiments. Galton thought of heredity quantitatively, and one of his chief principles

was the "law of regression," which states that children deviate from the mean of the population less than their parents deviate from the same mean. Such information would be of use to an insurance company, if the inherited character had anything to do with longevity, or to anyone actuarially interested for any other reason, but it did not help to understand the heredity of individuals.

With the coming of Mendelism, the statistical method of studying heredity gradually gave way to the individual method used in other organisms. Human characters, large numbers of them, have been traced through family histories. Though most of them are still somewhat less than clear, and many of them are rather obscure, they bid fair to fall into the same scheme as the inherited qualities of other organisms. No fact of human heredity is yet known which contradicts that scheme in any particular, and many such facts plainly support it.

Practical Applications.—Practical applications of knowledge of inheritance in man are being attempted. The eugenics movement dates from the work of Galton, who founded a professorship in that subject in the University of London. Strong organizations in several European and American countries are furthering this work. Such work in America centers largely in the Eugenics Record Office on Long Island, and in the Eugenics Research Association. The success of the whole movement depends first on accumulating a much larger fund of information concerning human heredity and second on a consummate wisdom in its use.

Much more advanced is the application of laws of heredity to such economic ends as the improvement of domestic animals and farm crops. The work in this field constitutes a sizable industry, largely under federal and state management, for the benefit not only of agriculture but of all who use its products.

CHAPTER II

FUNDAMENTAL STRUCTURE OF ORGANISMS

The ultimate interest of the student of heredity concerns the qualities of whole organisms. Yet to understand those qualities, he must know the manner in which the organisms are constituted. Rightly understood, the axiom that a whole is equal to the sum of its parts is as true in biology as it is in mathematics. Many of the most important clues to the workings of heredity are furnished by the facts of structure.

In his search for the physical basis of heredity the geneticist must look far deeper than ordinary anatomy. Organs and tissues are of little service, except occasionally in throwing light on the mechanics of development. The cells of which these grosser structures are composed are the largest units with which genetics must usually deal. Even the cells are too large and inclusive to be of prime significance in most problems of heredity; it is their minute components that are most revealing. Some of these components are readily visible with a microscope, but the most important ones are near or beyond the limits of visibility even with such optical aid.

Ubiquity of Cells.—The importance of cells to the determination of qualities of organisms rests partly on their pervasiveness. They are everywhere. Plants are composed of them throughout roots, stem, leaves, flowers, and fruit. Every tissue of animals is built of cells, and every activity is traceable to them. Muscle, bone, skin, glands, all are cellular. Blood, though largely liquid, contains hosts of cells which determine some of the most important properties of the circulating medium. Coordination of the many activities of an animal depends upon conduction of impulses by cells in the nerves, and mental processes depend upon brain cells. Even such dead structures as hair and nails are made of cells that were once alive. All development from seed or egg involves cells, and every stage, young or old, of plant or animal, is composed of these structural units.

Whatever inherited quality is under scrutiny, cells have made it what it is. And no matter what phase of hereditary transmission is examined, cells are found to be guiding and effecting it.

The Nature of Cells.—These cells are composed of a material known as protoplasm, nearly colorless, of a jellylike consistency, and comprising a mixture of proteins, carbohydrates, fats, lipoids, salts, and water. Of the several classes of organic compounds named, the proteins are most important in heredity. The proteins are built up of 1 or more of the 18 amino acids, in various proportions and arrangements. Because of this chemical structure, proteins are very complicated; they may be and are, therefore, of very many different kinds. Moreover, in their chemical relations proteins are very specific, that is, they do certain things with great precision and other things not at all. These two properties of proteins, their great specificity and their complexity of structure, are what make them so significant in living things. Because of them organisms can be of an enormous number of different kinds and can maintain these different kinds with a high degree of persistence.

Within a cell, the organic components exhibit a pattern; some of it is visible, some of it merely inferred. Somewhere in the midst of its protoplasm is almost always a *nucleus* (Fig. 5), usually rounded in form, perceptible in its natural state and readily made more so by any of the common dyes. The important component of the nucleus is a quantity of scattered protein material of a variety of kinds, collectively called the *chromatin.* As its name suggests, the chromatin is readily stainable, and it is this material which renders the nucleus conspicuous in dyed tissues. The nucleus also contains a liquid, in which the chromatin floats; there is often another body called the *nucleolus* in this fluid; and the whole is marked off from the protoplasm outside by a thin membrane whose tension tends to keep the nucleus round. Any substances which enter into commerce between the nucleus and the surrounding protoplasm must pass through this membrane.

In the outer protoplasm, called *cytoplasm,* are usually other structures. Frequently, especially in plants, there are *plastids.* These are oblong bodies, colored green as in leaves, red or yellow as in some fruits and flowers, of other colors or colorless in various other situations. Some plastids are engaged in supplying

nutrition, others in storing it, and some are of uncertain function. Many cells also contain *vacuoles*, droplets of enclosed liquids, having various uses or being merely incidental. There are probably always, also, rodlike, threadlike, or granular bodies of mainly lipoid composition, known as *mitochondria* or *chondriosomes*. An object similar to the mitochondria in composition, but having variable form and usually located near the nucleus, is

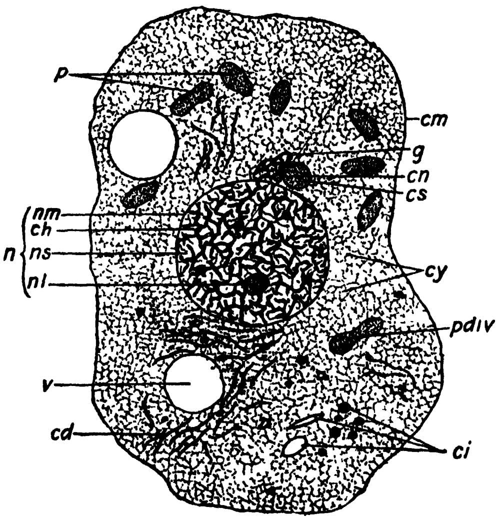


FIG. 5.—A typical cell. *cd*, mitochondria; *ch*, chromatin; *ci*, lifeless inclusions; *cm*, cell membrane; *cn*, centriole; *cs*, centrosphere; *cy*, cytoplasm; *g*, Golgi apparatus; *n*, nucleus; *nl*, nucleolus; *nm*, nuclear membrane; *ns*, nuclear sap; *p*, plastids; *pdiv*, plastid dividing; *v*, vacuole. (*From Shull, LaRue and Ruthven, Principles of Animal Biology, McGraw-Hill Book Company, Inc.*)

the *Golgi body*. Finally, in many cells there are lifeless objects, either produced by the cells themselves, such as starch grains or fat particles, or taken in from the outside and having no real part in the activities of the cells.

Of the cell structures mentioned, the chromatin of the nucleus is by far the most important in heredity. Reasons for this conclusion and details concerning the relations of the chromatin to genetic processes are presented in many places in later chapters. Of the cell contents outside the nucleus, the plastids have an important relation to the heredity of color in plants, on a basis very different from that of the chromatin. Some biologists have also suggested that mitochondria have a function in heredity, but there is little to indicate any specific connection of this sort.

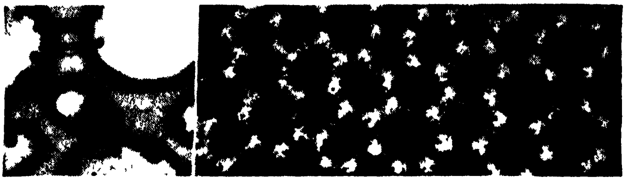
Partnership of Cells.—That the qualities of an organism depend on the cells in it would be assumed, merely from the presence of these structural units in all of its parts. The correctness of this assumption is rendered certain by knowledge of the way in which the organism is built in the first place and of the manner of its operation afterwards. It is not desirable to describe here the various physiological processes entering into embryonic development or into the processes of maintenance after that development is complete. One feature of these activities is, however, so general and so characteristic of the more minute mechanism of heredity, that it deserves emphasis. That general feature is the cooperation that exists everywhere among cells.

No cell works by itself in a multicellular animal. Every cell is influenced by others around it or even at a distance from it. This is a truism in physiology in general, but needs repetition with respect to anatomical features and to those physiological and psychological features in which there are known hereditary differences. In embryonic development a great deal depends on how the cells are placed relative to one another. A chain of events leads normally to a certain end result, but, if any part of the mass of cells is artificially misplaced, the succeeding changes may be profoundly modified (page 52). Place a section of the early nervous system anywhere else than in the middle of the back, and a double monster may result. Snip the eyestalk, and the crystalline lens may fail entirely. In later stages the growth of bones responds to pressure such as is exerted by adjoining bones; their internal structure is gradually braced against that pressure. Mental qualities whose variations are inherited depend largely, not on individual brain cells, but on how those cells are joined to one another. Probably nowhere in multicellular animals does anything of importance depend solely on the nature of individual cells, but it does depend in part upon interrelations among cells. This does not prevent the cells, however, from being in considerable measure responsible for the relationships that have been established.

How cells influence one another is partly unknown. Among tissue cells there are occasionally protoplasmic bridges (Fig. 6) from cell to cell, through which it is assumed that some sort of communication is effected. Structural bridges are not necessary, however, for dissolved substances can pass through any

of the ordinary envelopes around cells; and even the subtler forms of communication, such as waves of electric potential, can traverse such boundaries.

Germ and Somatic Cells.—In the many-celled animals there are two classes of cells, fundamentally different from each other in their relation to heredity. The somatic or body cells are directly responsible for the actual manifestation of inherited characters but have nothing to do with transmission of them except in primitive types of reproduction. The germ cells, on



a *b*

FIG. 6.—Cell bridges, protoplasmic connections between cells; *a*, in persimmon, note fine lines traversing heavy cell walls; *b*, in moss. (*After C. J. Chamberlain and B. Němec, from Seifriz, Protoplasm, McGraw-Hill Book Company, Inc.*)

the contrary, have everything to do with transmission from generation to generation in the higher forms of reproduction but are not at all directly responsible for the expression of most of the characters inherited.

The two kinds of cells are connected in animals in a very important unilateral genetic way. The germ cells of animals give rise in every generation to somatic cells, as well as to more germ cells. But the somatic cells do not produce germ cells, or do so only very rarely, in animals. The germ cells thus constitute a reserve out of which the genetic continuity of the germ cells and the repetition of the production of bodies in each generation are maintained. This sharp distinction between the two classes of cells does not obtain in plants; and there are biologists who believe it is not very sharp in animals. Where the unilateral genetic relation exists, however, it has very important consequences in the theory of heredity and deserves emphasis even if far from being universal.

CHAPTER III

PRODUCTION OF NEW CELLS

The process by which new cells are produced, always out of old ones, contains the key to two groups of facts which are very significant in heredity. First, it shows why all the cells of a multicellular organism must be expected to be genetically alike, at least in their beginnings. Second, it discloses some features

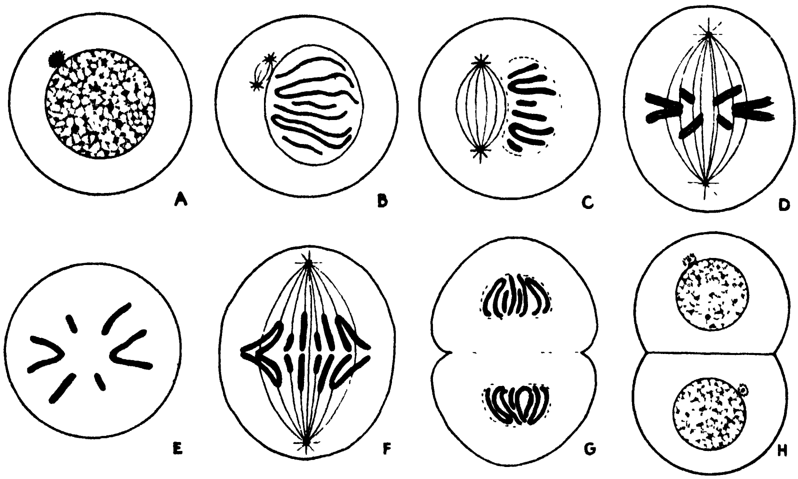


FIG. 7.—Cell division. *A*, cell not dividing, ordinary nucleus with divided centriole at its upper left; *B*, chromatin of nucleus resolved into chromosomes, spindle forming at left; *C*, nuclear membrane dissolving, spindle enlarged; *D*, duplication of chromosomes, now located on middle of spindle; *E*, same stage as *D*, but viewed from end of spindle so duplication of chromosomes is not seen, *F*, duplicated chromosomes moving or being drawn to opposite ends of the spindle; *G*, chromosomes beginning to form nuclei, cell body beginning to divide, *H*, two cells completely reconstructed.

of the constitution of cells which even the closest examination of them at other times than their birth does not reveal—features which bear very directly on the operations of heredity.

Scheme of Cell Duplication.—The general procedure in the production of new cells in animals is as follows. The chromatin in the nucleus, which at other times appears scattered, becomes

assembled into several strands or ropes of various lengths, which are called *chromosomes* (Fig. 7, *B*). Outside the nucleus there is often a minute dotlike object, the centriole, which divides into two. Its two parts move away from one another, and fine lines form between them and around them, making a spindle-shaped figure with a star at each end. This spindle lies beside the nucleus, whose surrounding membrane now is dissolved away, allowing the chromosomes to move over into the midst of the fibers of the spindle (Fig. 7, *C*).

About this time each chromosome is seen to be double (*D*), i.e., each chromosome has produced a new chromosome which,

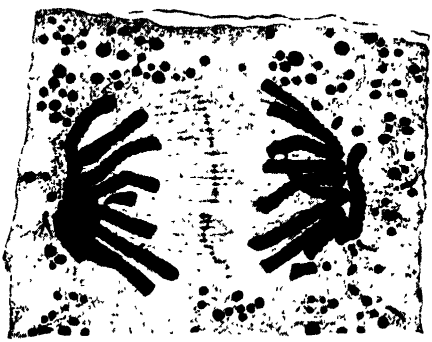


FIG. 8.—Formation of dividing wall in plant cell. (*From W. Robyns, in Seifriz, Protoplasm, McGraw-Hill Book Company, Inc.*)

as is pointed out later, is an exact replica of itself. These identical chromosomes are then drawn apart, as if being pulled apart by some of the fibers of the spindle (*F*). One of the two similar chromosomes produced by each duplication process goes to each end of the spindle, so that two groups of chromosomes, identical with one another and with the original chromosomes, are placed at opposite poles of the cell (*G*).

Here each group becomes surrounded by a new nuclear membrane, the chromosomes begin to become diffuse again, and the body of the cell becomes furrowed, then completely divided, between the new nuclei (*H*). Two new cells have replaced the old one.

Duplication of plant cells usually differs from that just described in two unimportant respects. First, the flowering plants do not have centrioles; yet spindles are formed and they operate in the same general way as in animals. Second, in the

division of the cell body between the new nuclei, there is no furrow from the outside. Instead, a row of pellets is deposited across the middle of the cell (Fig. 8). These knots, as they grow, merge and form a wall that separates the two new cells.

Finer Constitution of Chromosomes.—While the account just given describes cell division as it appears on casual observation, it contains little more than a hint as to the real meaning of that process. To grasp its importance, one must know the minute architecture of the chromosomes. Fortunately, this finer construction may be observed in favorable types of cells and is to be inferred in others that do not reveal their organization so directly.

Each chromosome is made up of a chain of minute bodies (the *chromomeres*) strung like beads on a fine thread or imbedded like boulders in a very slender stream. Some of these beads can be seen when the chromosomes are long and exceedingly slender threads, as they are when the first condensation of the chromatin takes place preparatory to cell division. The chromosomes are greatly extended then, and the nodules are relatively far apart, so that they are separately visible. The visible beads on these strings are of many different sizes (Fig. 9), and there are sound reasons for believing that many others, probably also of different sizes, are too small to be seen. Presumably, also, they have different chemical compositions, which is more important. Though some of these little pellets may be just like others in the same chromosome, they have been proved to be in the main different from one another.

Pattern in the Chromosomes.—These motley particles have a definite arrangement in their respective chromosomes. To make the portrayal concrete, picture the first one at the end of a chromosome as a large one, followed by several of minute but different sizes, then one of medium volume. After this several small ones, two large ones in close succession, a few small ones, then a medium and several small ones, again a medium and a large one; and so on. An actual pattern, somewhat different from the hypothetical one just described, is shown in Fig. 9. While there may be only several dozen visible chromomeres in a given chromosome, their total number, including those too small to see, must usually amount to hundreds. All of them, there is good reason to assume, have as definite a pattern of arrangement as do those which can be seen.

Now, every cell of an organism has in it a chromosome of the same design. Particle by particle, from one end of the chromosome to the other, the same order of sizes is followed in one chromosome of every cell. Indeed, there are usually two chromosomes having this same pattern in every cell of the same individual. Moreover, this same arrangement of particles is found, with minor modifications, in every individual of the same species. In Fig. 9, each pair of chromosomes shown came from a different cell, even from a different individual. The similarity of their patterns is indicated by the dotted lines passing through

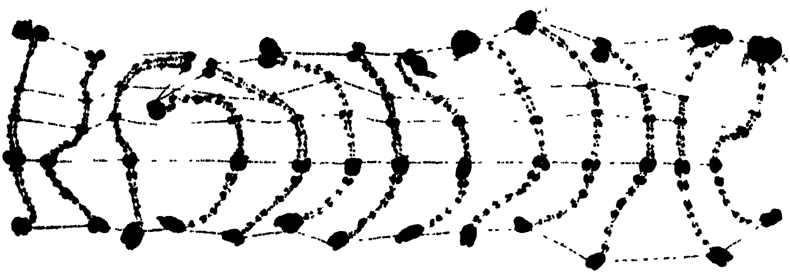


FIG. 9.—Pattern of chromomeres of different sizes in one of the chromosomes of a grasshopper. The chromosomes are in pairs, from thirteen different individuals. The horizontal dotted lines pass through homologous chromomeres. (*Modified from Wenrich*)

corresponding nodules. In some situations there are even three chromosomes that have the same pattern in each cell of the species, as in the endosperm of seeds, but usually there are but two equivalent chromosomes per cell.

Individuality of Chromosomes.—All the other chromosomes in the cells are different from these two. Two of them are alike in their pattern of beads, but different from all the rest. Two more are alike in pattern, but it is a different pattern from that of any other two chromosomes. In this manner, each chromosome is twin to another. Each chromosome is like one other, but different from the remaining ones. In the chromosomes of the onion (Fig. 10) this likeness of the chromosomes two by two is observable, not so much in their patterns of nodules of different sizes as in their lengths, some being long, some medium, some short.

The chromosomes thus possess an individuality, which, however, is shared between twins. They could very well be named, just as people are named. All of the chromosomes having a

given pattern of their constituent chromomeres might be called A, whether in one cell or another, and no matter in what individual of the species. A chromosome of another pattern could be designated B, wherever it occurred, in different cells or different individuals. A third pattern might be called C, and so on. Thus, the chromosomes of a cell, of an individual, or of a species, would consist of two A's, two B's, two C's, and so forth.

It is possible that some of the minute globules that are strung along chromosome A may be identical with some in chromo-

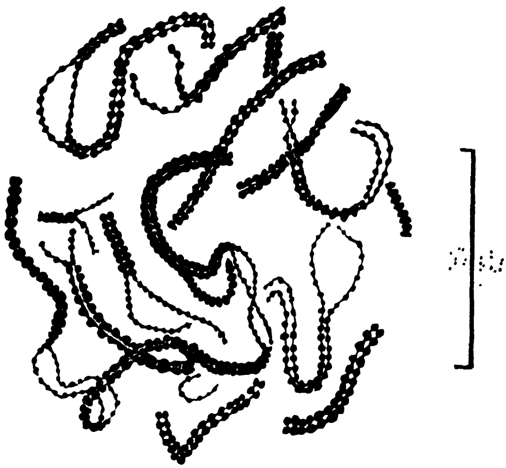


FIG. 10.—Chromosomes of the onion, whose individuality is observable in their size (number of chromomeres) rather than in patterns of chromomeres of different sizes. (*From Belling, Univ. Calif. Publ. Bot.* 16.)

somes B or C. Indeed, from a consideration of the way in which chromosomes have evolved, it is to be expected that there are some such repetitions. That is not, however, an important fact for the present. Since a given particle in one position has been shown to have an effect different from that of the same particle in another position, the chromosomes may be regarded as wholly different except as between twin chromosomes.

The two chromosomes that are alike are called homologous chromosomes from the fact that they must have evolved from the same source. Two A's are homologous with one another, even if they come from different individuals; two B's are homologous; and so on.

Significance of Cell Division.—The importance of the method by which a cell divides should now be apparent. When the

chromosomes are doubled, each nodule which the chromosomes contain is duplicated. Though this statement places observation before inference, it probably reverses actual cause and effect; for it is presumably the duplication of the single beads which brings about the doubling of the whole string. Since the nodules are of different sizes and different chemical compositions from end to end of each chromosome, only a duplication of each pellet would effect a genuine duplication of the chromosome. Any other method would result in the formation of two unlike new chromosomes out of the old one. Furthermore, since the chromosomes in each cell differ among themselves (except homologous chromosomes), any other method than the one actually employed, of duplicating each chromosome and carrying one of the resulting two daughter chromosomes to each of the new cells at each division, would yield two dissimilar cells instead of two alike.

The value of this duplication, part by part, in maintaining a combination of living structures which is successful—which had to be successful in order to survive—can hardly be overestimated. Any scheme of cell division which freely permitted one cell to be different from the other cell arising at the same division would lead to wasteful chaos. Insofar as this regularity is maintained, every cell in a multicellular organism ought to have the same constitution as every other cell in the same individual. That is, muscle, nerve, bone, gland, and blood cells should be originally fundamentally alike. Whether they do maintain this similarity indefinitely has been debated, but cell division does not appear to be an agency by which they might become different.

This is not to say that irregularities never creep into cell division. Occasionally, after a chromosome has become duplicated, both of its daughter chromosomes go to the same cell, while the other cell lacks a chromosome of that identical constitution. Such failure of chromosome duplicates to go to different cells is called *nondisjunction;* certain special genetic phenomena are dependent upon it.

Division of Plastids.—Since some of the inherited characters of plants, such as variegation of leaves, are due to the plastids, it is worth while to point out that these structures are autonomous in their multiplication. That is, their increase in numbers is not in any way dependent on the duplication of chromosomes and has no necessary connection with the process of cell division.

Plastids multiply by simply dividing in two, in a manner analogous to vegetative or asexual reproduction of simple organisms, described in the next chapter. This division results in two identical plastids each time, but the rest of the cell is in no way involved. Plastids may go on dividing when the cell as a whole, including its chromosomes, is not dividing; and plastids do not have to divide when the cell divides. At cell division, some of the plastids are located in one daughter cell, some in the other. If the plastids are of two or more kinds, there is consequently no assurance that the two cells produced by cell division will be equivalent with respect to them. In this feature they differ from the chromosomes.

CHAPTER IV

ORIGIN OF NEW INDIVIDUALS

Since heredity involves continuity between generations, the manner in which new individuals are produced must exercise a fundamental influence upon the nature of transmission. The several modes of reproduction differ greatly with respect to their bearing on inheritance. The differences relate mostly to their effect on the stability of the species. Some forms of reproduction tend to preserve, unchanged and alike, all individuals belonging to a given line of descent. Other forms contribute materially

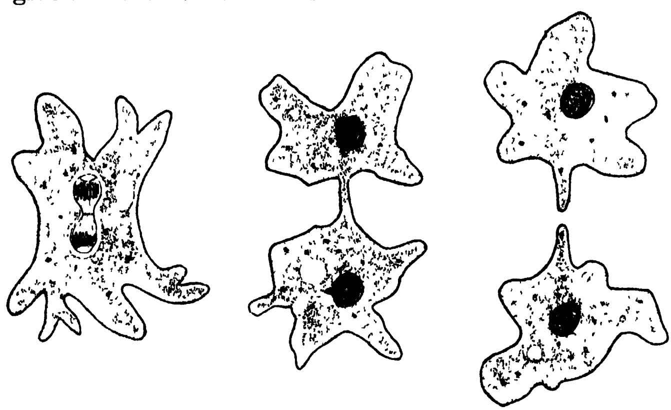


FIG. 11.—Fission, or asexual reproduction, in Amoeba. The chromosomes shown in the first figure suggest that the division is a qualitatively equal one.

to the variability of the species, that is, to the differences among individuals, even among those closely related. The proneness to constancy or the tendency to change is the chief feature of a mode of reproduction which concerns the student of heredity; all other elements of the process are minor.

Asexual or Vegetative Reproduction.—Any form of reproduction in which a considerable fraction of the parent goes over into each of the offspring offers an excellent opportunity for the individuals of both generations to be similar. *Fission*, or the nearly equal division of the parent, is employed by many of the unicellular organisms. In Amoeba (Fig. 11) the cell simply

divides into two. Though the fine details of the process are not easily observed, the nucleus of Amoeba is resolved into minute bodies which may be likened to chromosomes. If these bodies are duplicated during fission, then the two amoebas produced should be as much alike as are the two cells resulting from cell division in multicellular organisms. In Paramecium, there are two nuclei, a large one and a small one. In fission, the small nucleus forms minute chromosome-like objects, but the large one does not (Fig. 12). However, the large nucleus is not very important in this connection since it periodically disintegrates and is replaced by pieces of the small one. Consequently, in the long run, fission in Paramecium is of a type which appears to provide equality of the two cells produced.

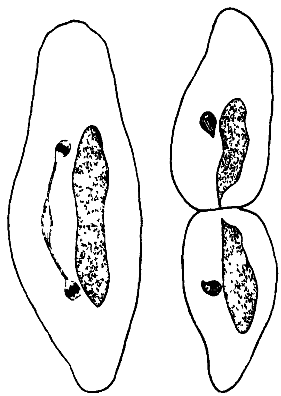


FIG. 12.—Fission in Paramecium. Only the small nucleus has any long-term importance in reproduction. Its chromosomes indicate that the division is probably a qualitatively equal one.

When the division of the parent is very unequal, reproduction is usually called *budding*. This name is particularly appropriate when a small portion of the parent must first be protruded before it is pinched off. Hydra commonly reproduces in this way. The body wall of this animal consists of two layers of cells, and when a new individual is to be produced this wall is simply elevated like a hollow haycock. The protrusion is extended (Fig. 13), develops subsidiary protrusions for tentacles, opens a mouth among the bases of the tentacles, and then is pinched off. Since the cells which enter into the bud have been produced by the duplicating method of cell division described in the preceding chapter, they are presumably like all other cells in the body. Even if they have developed some features not like those in cells in the upper part of the body, they should nevertheless be genetically like all the other cells. Consequently, the offspring would be expected to be identical with the parent.

In some forms of asexual reproduction, small groups of cells (variously called *gemmules* or *statoblasts*) are separated off from the body. Each group is usually enclosed in a resistant capsule of some sort which protects it until conditions favorable to

growth prevail. Then the capsule is thrown off, and the enclosed cells are transformed into a new individual. In some organisms single cells are thus set aside for reproduction. These cells are generally called *spores*. Whether these types of reproduction conduce to similarity among individuals depends on the nature of the divisions by which the reproductive cells are produced. Gemmules and statoblasts give every sign of leading to stability;

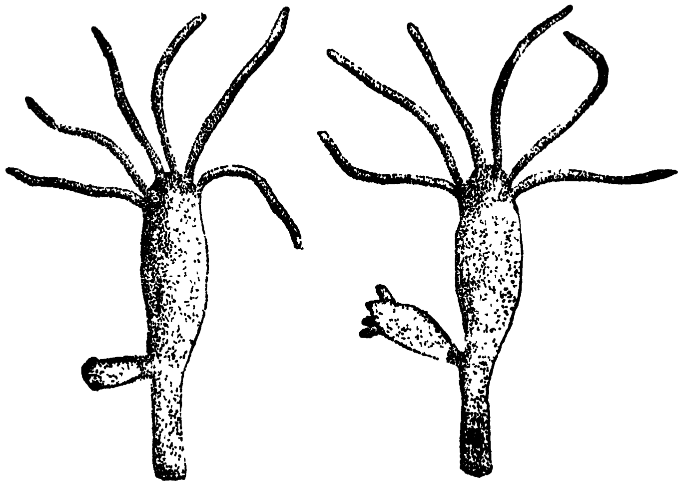


FIG. 13.—Asexual reproduction in Hydra; two successive stages of budding.

spores in plants may lead to variability for reasons similar to those which entail variation in biparental reproduction (see below).

Variability and Asexual Reproduction.—Though it is usually expected that no difference between parents and offspring will arise in asexual reproduction, that expectation is based chiefly on the fact that vegetative reproduction stems originally from what seems to be ordinary cell division, and such division appears to result in equal cells. What other evidence is there relating to this anticipated stability? Certain unicellular organisms have been bred for many generations and watched for changes. Very few have occurred. In some such investigations no modification whatever has been detected. In a few studies, new types of individuals have occasionally arisen.

Do these occasional modifications indicate that, after all, asexual reproduction is not the conservative process it seems to be? Probably not. So far as the qualities of the organisms are

determined by their chromatin, the changes are presumably of the nature of mutations, which constitute the basis of much of evolution. These mutations do not depend on the type of reproduction. They are changes which would occur in some individuals whether they reproduced or not. In other words, reproduction is not causing them.

If any differences were displayed in the cytoplasm, in structural or physiological units which multiply autonomously and maintain their characteristics independently of influence of the chromatin,

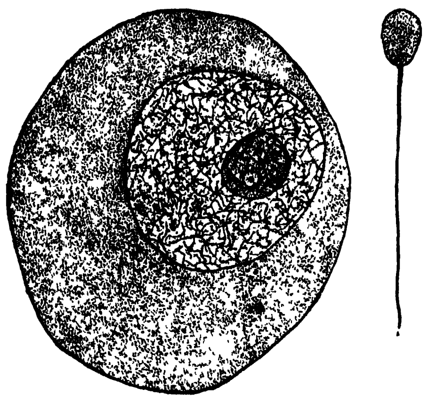


FIG. 14.—Typical germ cells. *A*, egg of starfish magnified × 500; *B*, spermatozoon of rabbit magnified × 4000.

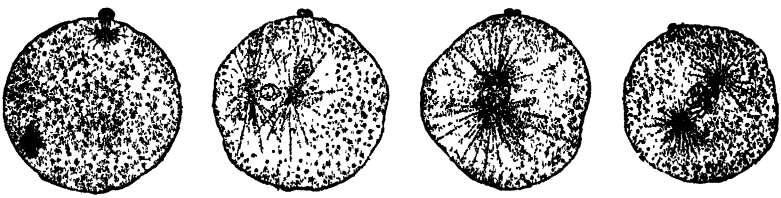
an irregular segregation of these should take place, and dissimilar lines should arise, in asexual reproduction. Aside from plastids (page 32), there is little evidence of the existence of such autonomous units, and differences dependent on other things have usually proved temporary.

Biparental Reproduction in Higher Animals.—In typical sexual reproduction two parents are involved. These parents are differentiated in the higher animals into sexes which are structurally different, not only with respect to the reproductive organs but in other ways as well. Both sexes in the higher animals produce germ cells. In the females these germ cells are relatively large and passive, and are called *eggs*. In the males the germ cells are very small and actively motile, and are called *spermatozoa* (or sperm). Typical germ cells are shown in Fig. 14, at very different magnifications.

Egg and spermatozoon are in some manner brought together, and they merge into a single cell, cytoplasm with cytoplasm, nucleus with nucleus. This union is known as *fertilization* (Fig. 15).

From the fertilized egg, by a series of cell divisions and changes of shape, described in the next chapter, a new individual is produced.

What Is an Egg?—Since in some forms of asexual reproduction the new individual starts from a single cell, a spore, it is important to know the distinguishing marks of germ cells in sexual reproduction. The spermatozoa would never be mistaken, for they have



A *B* *C* *D*

FIG. 15.—Fertilization of an egg. *A*, entrance of spermatozoon at lower left; *B*, the egg nucleus and sperm nucleus approach one another; *C*, the two nuclei are side by side; *D*, the nuclei have merged, and their chromatin is being resolved into chromosomes preparatory to the first division of the fertilized egg. The figures also represent meiosis or maturation; at the top are the two small cells produced in the two meiotic divisions.

a peculiar form not imitated by any other kind of cell, though that is not their real distinction. The eggs, however, have a shape that many other cells take. Hence concerning them the question may often arise whether they are eggs, and if so, what makes them such.

The crucial feature of both egg and sperm in animals is that the cell has undergone a process known as *meiosis* or *maturation*. In typical meiosis the cell experiences two rapidly succeeding divisions. The two cells produced at each division are very unequal in size in the case of eggs, one being very small. Maturation of an egg is represented along with fertilization in Fig. 15. In *A* of that figure, one of the two divisions has taken place, the small cell being shown at the top. In *B*, both divisions have been completed, and there are two small cells at the top.

In the course of these meiotic divisions the number of chromosomes is reduced to half. The final egg or spermatozoon has only one chromosome from each set of chromosome twins. This is an

exceedingly important feature of a true germ cell. How the chromosome number comes to be thus reduced is described more fully in a later chapter.

Biparental Reproduction in Higher Plants.—In flowering plants the male and female germ cells are often produced on the same plant, commonly in the same flower. Such a flower is diagram-

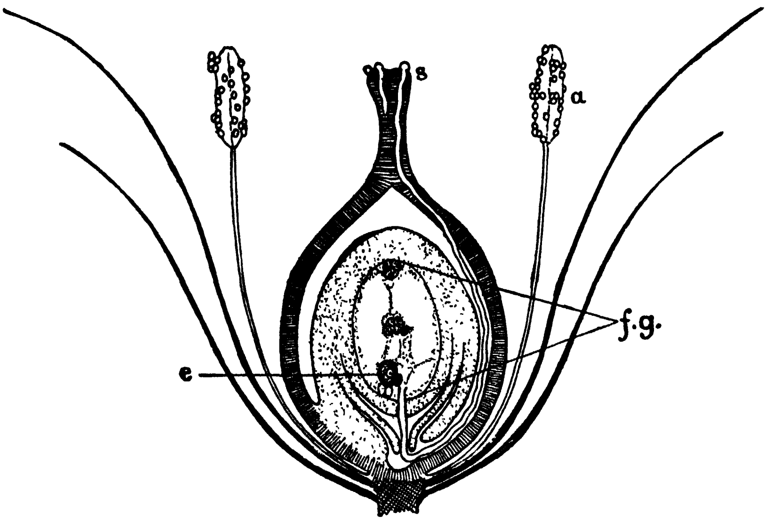


FIG. 16.—Diagrammatic section through a flower; *a*, anther; *e*, egg; *f.g.*, embryo sac; *s*, stigma. The white channel down from the stigma is a pollen tube. One male nucleus is the dark body at the right of the egg, the other male nucleus is at the right of two other nuclei in the middle of the embryo sac. (*From Sinnott and Dunn, Principles of Genetics, McGraw-Hill Book Company, Inc.*)

matically represented in Fig. 16. The egg is one of eight cells forming an ellipsoidal mass (the embryo sac, *f.g.*), each cell of which has only the reduced number of chromosomes. That is, what corresponds to meiosis occurs early, and the reduced cell then divides until eight cells are formed, one of them being the egg (*e*).

The male cells in flowering plants are produced in the anthers (*a*). The entire pollen grain is composed of cells which have the reduced number of chromosomes. Two of these reduced cells are male cells. To function in reproduction, the pollen must fall on the stigma (*s*) of some flower and develop a tube (Fig. 17) down through the stigma, style, and other tissues until it reaches the egg. The two male cells pass down this tube, usually near

its growing tip. One of them fertilizes the egg, and from the combined cell an embryo develops. The other male cell unites with usually two other cells near the egg (in the middle of the embryo sac), and the triple product forms the endosperm or nutritive part of the seed. These two fertilizations have already taken place in Fig. 16. When the seed germinates the embryo feeds on the endosperm until it can begin to secure its nutrition from the air and soil.

Biparental Reproduction in Protozoa.—With this outline of sexual reproduction in the more complex animals and plants

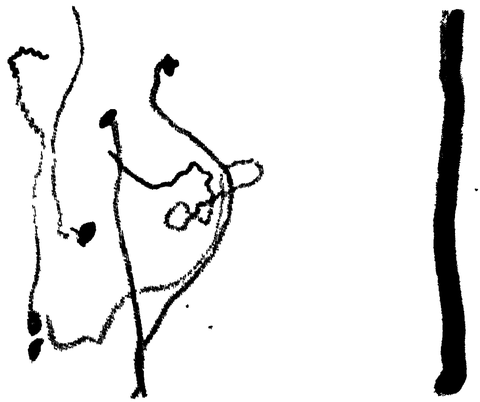


FIG. 17.—Growth of pollen tubes. Left, pollen germination on artificial medium. Right, tip of one pollen tube, with tube nucleus followed by the two male nuclei. (*Courtesy of General Biological Supply Company.*)

in mind, it will be possible to understand the corresponding processes in unicellular organisms more readily. Paramecium must suffice as an example of sexual reproduction in the protozoa; most of these simple animals probably employ this method along with the asexual one already described.

The really sexual part of this reproduction consists of *conjugation*, in which two paramecia come together side by side (Fig. 18). These two individuals are alike, that is, they are not distinguishable as male and female. Of the internal events, only those concerning the small nucleus are of importance. This nucleus divides several times, part of the process resembling the two divisions which constitute meiosis in higher animals. What happens to the nucleus during these divisions cannot be observed in detail. Whether there are pairs of homologous chromo-

somes which become separated into different nuclei, as happens in more complex organisms, cannot be directly ascertained; but the genetic results of breeding experiments with Paramecium indicate that such separation does occur (Raffel 1933; Sonneborn and Lynch 1934).

As a result of the series of divisions of the small nucleus, each of the conjugating paramecia has two small nuclei; the others produced by these divisions degenerate. The two surviving nuclei are slightly different (Fig. 18), and one may be regarded as male, the other as female. The male nucleus of each individual creeps through the now nearly fluid walls of the animals into the other individual and there fuses with the female nucleus.

By the repeated division of this combination nucleus and the division of the body as a whole, offspring are produced which descend from both of the conjugating paramecia.

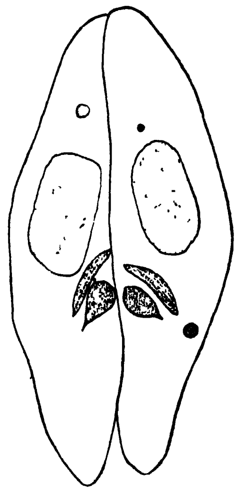


FIG. 18.—Conjugation in Paramecium. The spindle-shaped small nucleus just below the middle of each individual migrates into the other animal and fuses with the more rounded small nucleus there. From the combination nucleus the nuclei of subsequent generations are derived.

Variation and Sexual Reproduction.—As was indicated earlier in this chapter, sexual reproduction leads to differences among the offspring and differences between them and their parents. This is true wherever such reproduction occurs, whether in complex animals, flowering plants, protozoa, or any other organisms.

The reason for this variability lies in the events of meiosis. The germ cells when mature have only half of the chromosomes of the individual which produces the germ cells. In making up this half-group of chromosomes, one member is drawn from each of the pairs of homologous chromosomes. Now, if the paired chromosomes were identical in their composition, the reduction in the number of chromosomes by this very regular selective method would not result in variability. All germ cells of one individual would be alike. But the chromosomes need not be identical. In animals they are very seldom entirely alike, and in plants they are often different in some respect. Homologous chromosomes are similar but not usually identical.

Under these circumstances, drawing one chromosome of each pair for inclusion in a germ cell gives room for much variation in the various cells. The nature of a germ cell depends on which of the two chromosomes it has received from each pair. Differences among the germ cells lead to differences among the offspring and to differences between offspring and parent.

Parthenogenesis.—In some animals and plants an egg may develop into a new individual without having first been fertilized

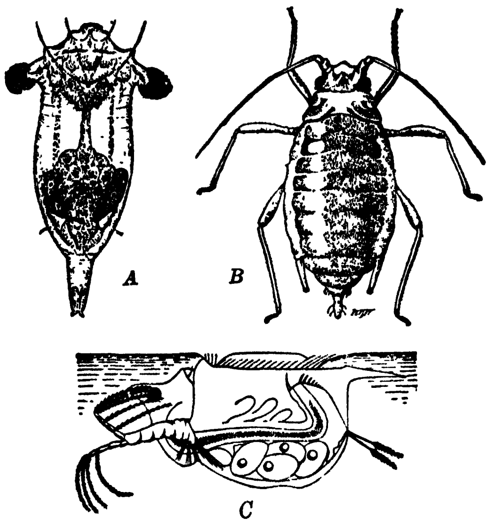


FIG. 19.—Parthenogenetic animals. *A*, a rotifer; *B*, an aphis or plant louse; *C*, a crustacean, suspended from the surface film of water, and showing several parthenogenetic eggs in the brood chamber below. (*A from Harring, Canadian Arctic Expedition; B from Webster, U. S. Department of Agriculture; C after Storch in Schulze's Biologie der Tiere Deutschlands.*)

by a spermatozoon. Such development is known as *parthenogenesis*. Several groups of animals employ this method as their principal mode of reproduction (Fig. 19), and in some species it is the only method known. Offspring produced in this way have only one parent, as do offspring produced asexually, so that many decades ago, when the nature of the process was not understood, animals now known to be parthenogenetic were said to reproduce asexually. However, the parent which reproduces parthenogenetically has a structure similar to that of typical females in related species, and the cell which develops into a new individual has undergone a process similar to typical maturation. Conse-

quently, the parent is regarded as a female, her reproductive cell as an egg, and the mode of reproduction as sexual even though uniparental.

Variation and Parthenogenesis.—Whether parthenogenesis results in different kinds of offspring from the same parent depends on the type of maturation which the egg has experienced. If the maturation is typical, in that it involves two cell divisions and the number of chromosomes is reduced to half, the eggs should be of various kinds, and the individuals produced from them should differ among themselves, much as in biparental reproduction. The eggs of the honeybee, ants, wasps, and many other similar insects undergo this typical maturation and display the expected variability.

In some other animals, however, the egg is produced as a result of only one maturation division, and the chromosomes are not reduced. There is no choice, therefore, among the chromosomes when the egg is constituted. Each egg receives a duplicate sample of every chromosome that the female herself possesses. The offspring derived from such an egg is genetically identical with the parent, and consequently identical with all other offspring of the same parent. It would be possible, if the chromosomes have some way of exchanging parts with one another, for variability to exist among these offspring; but such exchange has not been demonstrated in parthenogenetic females, and breeding tests in parthenogenetic species have generally shown that the individuals of one strain are all alike.

Limited Field Considered.—Since inheritance in man, most of the economic animals, and many cultivated plants is effected through biparental reproduction, that is the type that is emphasized in this book. It is worth while to know how much of the field of genetics is omitted when parthenogenesis and vegetative or asexual reproduction are relegated to a minor position.

CHAPTER V

DEVELOPMENT OF NEW INDIVIDUALS

Since most characters, in which organisms of the same species differ, and which thus come within the purview of the geneticist, appear only well along in the development of the individual, or even only in the adult, the steps by which that stage is reached are of some importance. Only those features of the process which have a real bearing on questions of organic determination can be emphasized here; but to understand them an elementary knowledge of the general course of development is essential. That hereditary qualities, represented in the germ cells only by exceedingly minute bodies not at all like the characters by which transmission must be judged, should unfold gradually during this development and appear as unerringly in the adult as they do is one of the marvels of living things.

Cleavage.—The fertilized egg divides into two cells, these two by simultaneous divisions into four, and so on by repeated divisions until hundreds of cells are produced (Fig. 20). All of the divisions involve chromosomes, in the typical process as described in Chap. III, and presumably all cells are genetically duplicates of the fertilized egg itself. The details of this cleavage vary in different species, depending on the amount of stored food the egg contains, on how closely the cells cling to one another, and on other things. Always, however, it leads to the formation of a hollow ball of cells, the *blastula* (Fig. 20).

Gastrulation.—The single layer of cells around the hollow of the blastula then becomes a double layer, typically by an indentation of the cells on one side. This inturning of cells is shown in Fig. 20 for the starfish, and in that animal does not proceed much farther than is there shown. In some other animals the indentation is broader, as in Fig. 21, *A*, and proceeds until the inturned cells lie against those of the opposite side of the embryo (*B*). The original hollow of the blastula has then disappeared. The two layers of cells are known, respectively, as

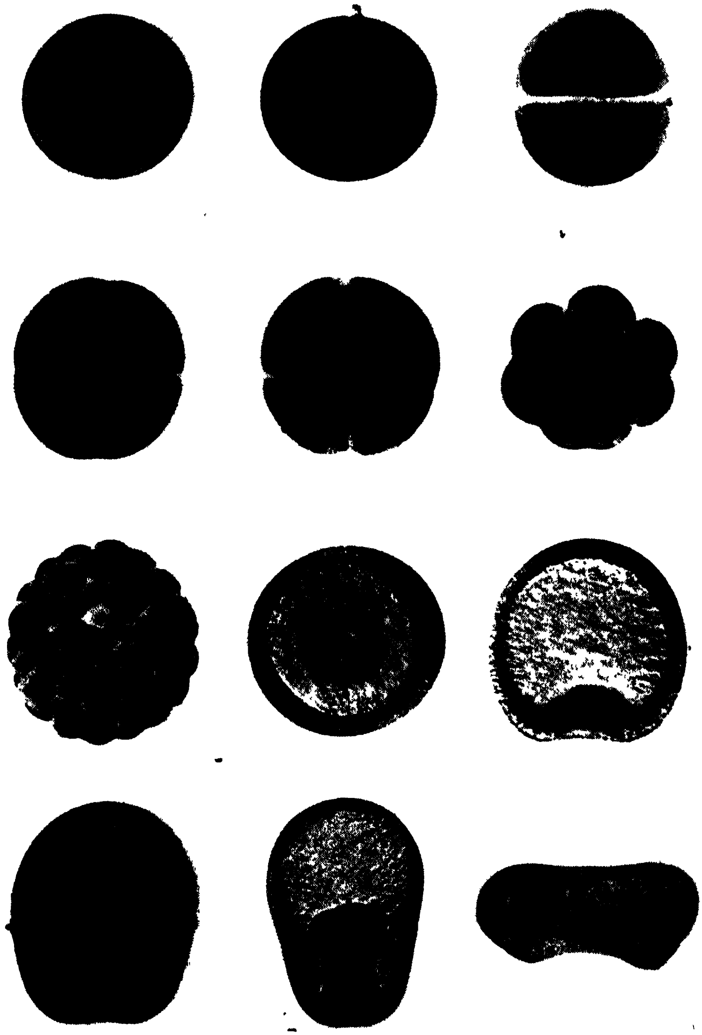


FIG. 20.—Development of the starfish. First figure, immature and unfertilized but fully grown egg (primary oöcyte); second, maturation stage, showing polar body; next five, successive cleavage stages; eighth, blastula; next three, steps in gastrulation; last figure, larva. (*Courtesy of General Biological Supply Company.*)

the *ectoderm* and *endoderm*, and this stage of development is called the *gastrula*.

Closely following gastrulation a third layer of cells, often poorly defined as a layer, is formed between the ectoderm

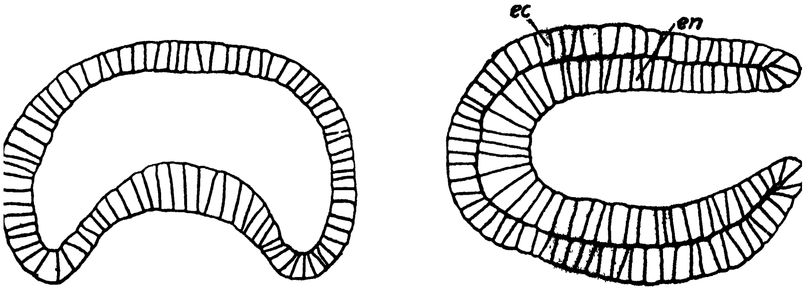


FIG. 21.—Gastrulation in Amphioxus; two stages, early (*A*) and late (*B*). *ec*, ectoderm; *en*, endoderm.

and endoderm. It is called the *mesoderm*. In some animals it is pinched off from the endoderm, which first rises in a ridge or fold, as in Fig. 22. In others the mesoderm is split off from the

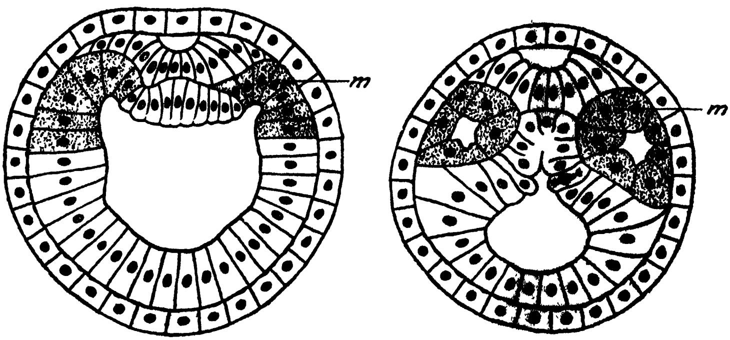


FIG. 22—Formation of mesoderm in Amphioxus. The shaded portion of the endoderm (*m*) is pushed out as a fold and subsequently pinched off to form the mesoderm.

endoderm by a mere separation of the cells into two groups, without first rising as a fold.

Ordinarily, from each of these three layers of cells, a definite group of organs is produced.

Organ Formation.—The manner of formation of organs out of these layers depends on the thickness and regularity of the layers. In the ectoderm and endoderm, which are often only one cell and

usually not more than three or four cells thick, most organs have their beginning in a bending or folding or protrusion of the layer. Only a few examples can be given.

The brain and spinal cord of vertebrate animals begin as a pair of folds elevated in the ectoderm on the outside of the embryo (Fig. 23). These folds come together and merge at the top, thus cutting off a tube beneath the ectoderm. This tube is gradually transformed into the central nervous system. The eyes begin as fingerlike protrusions from the side of the brain. The lens of the eye results from a thickening or folding of the ectoderm opposite this outgrowth from the brain. The inner division of the ear, which is the sound-perceptive part, sinks in from the ectoderm at the side of the head to form a pear-shaped sac.

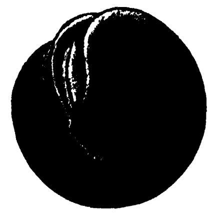


FIG. 23.—Central nervous system of frog being formed by folds of the ectoderm.

The endoderm, which from the beginning forms the digestive tract and later becomes the lining of it, folds or protrudes in a number of places to start the glands and other structures connected with the digestive tract. One of these glands is the liver (Fig. 24), which evaginates from the lower wall of the embryo intestine just behind the stomach. The bud thus produced branches in complex fashion by further evaginations as development proceeds. Other structures protruded from the digestive tract are the lungs and thyroid gland, each from a single outpocketing, and the pancreas which is derived from two such projections.

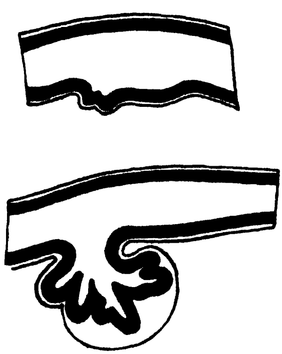


FIG. 24.—Two stages in the protrusion of the liver from the endoderm of the lower side of the intestine of the embryo.

All that the endoderm forms in these organs is the lining membrane. The remainder of them, which is bulkier by far, is furnished by the mesoderm. This layer is so thick and irregular in many places that bending or folding is not a feasible method of development. Instead, its cells merely creep into the spaces between the branching projections of the endoderm or rest passively while the endodermal protrusions grow out and branch among them. In the end these mesoderm cells are included in

the organ, so that much more of the liver, pancreas, or lungs is mesoderm than endoderm.

Development in Plants.—The fertilized egg in a flowering plant (Fig. 16) multiplies by repeated divisions and produces an embryo which is already recognizable as a minute plant in the seed (Fig. 25). This embryo develops no farther until the seed comes into conditions favorable to growth, which may be delayed for years. At that time the seed coats are burst, stored

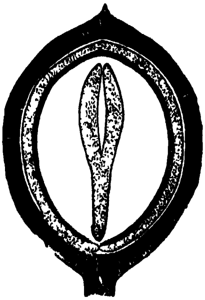


FIG. 25.

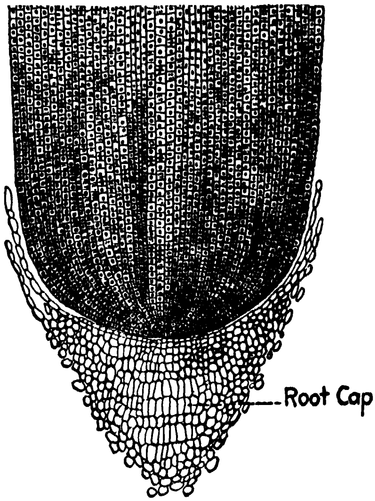


FIG. 26.

FIG. 25.—Ripe fruit of a flowering plant, including a seed. The Y-shaped stippled body in the center is the embryo. (*From Sinnott, Botany: Principles and Problems, McGraw-Hill Book Company, Inc.*)

FIG. 26.—Growing point in tip of root. Dividing cells here add to root cap ahead of advancing root, and to the root behind them, causing the advance. (*From Sinnott, Botany: Principles and Problems, McGraw-Hill Book Company, Inc.*)

food is utilized for growth, and typical root, stem, and leaves are soon produced.

Plants characteristically produce in their stems and roots certain types of tissues which are established by small groups or layers of cells which remain young or embryonic. There is such a group near the tip of each root (Fig. 26), which adds continuously to the protective root cap ahead of it and to the root itself behind it. Similar growing points provide continuous elongation of the stem and of lateral branches as well (Fig. 27). Stems and roots are made thicker by a layer of similar growing cells a little way in from the surface. These cells add to the differentiated tissues within and without their cylinder, but as a

developmental layer do not themselves increase in number except as the cylinder enlarges.

The differentiation in plants is much less specialized than in animals. A general scheme of structure pervades stem and root, which form the bulk of most plants. One part of a stem is very much like another part distant from it. Only in flower,

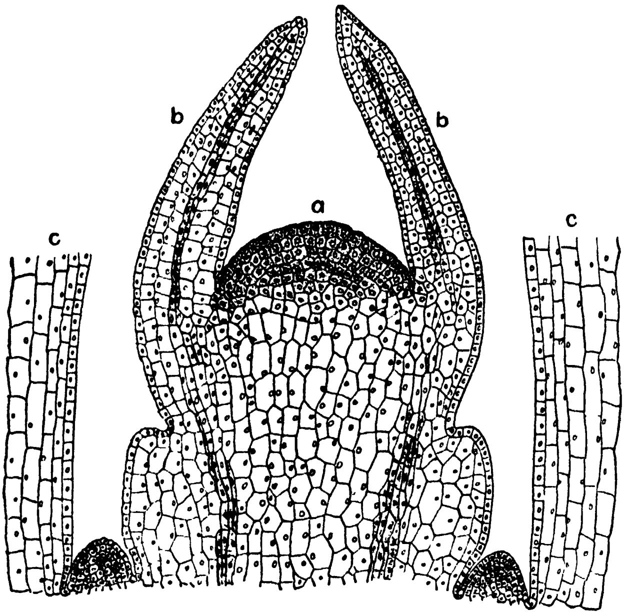
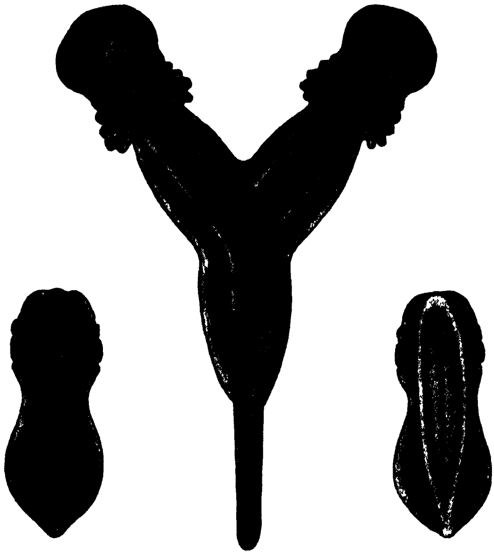


FIG. 27.—Growing point in stem (*a*). The projections at the sides (*b*, *c*) are leaves, the bulges just above the lower ones secondary growing points. (*From Sinnott, Botany Principles and Problems, McGraw-Hill Book Company, Inc.*)

fruit, and leaves is there strikingly different development, and these are repeated after the same design in many places.

Organic Determination.—The student of heredity is concerned with the process of development in two ways. The less important of these ways is that the characteristics from which he deduces the laws of heredity must usually pass through the developmental processes before he is aware of them; and in development there lies the possibility of changes which might obscure their true nature and confuse his judgment of them. A frog embryo, for example, normally has the form shown in Fig. 28, *A*, at an early stage. If a hair be tied around the furrow

between the first two cells produced from a cleaving egg, a double-headed monster (*B*) develops. Or if the egg is placed in a solution of cane sugar, the ridges that form the central nervous system fail to come together (*C*). These are things that would not ordinarily happen in nature, but they illustrate how environmental differences may disturb the end result and obscure the workings of heredity.



A B C

FIG. 28.—Environmental modification of embryos. *A*, normal embryo of frog; *B*, double-headed embryo due to girdle around furrow in 2-cell stage; *C*, failure of folds of nervous system to meet, owing to cane sugar solution.

The much more important relation of development to heredity is its embodiment of what may be called organic determination. What causes one part of an organism to become one thing, another part something else? Ultimately, the heredity units or *genes* must be responsible, since one egg becomes a starfish, another a snail, in the same sea water. Other things in the cells may be *immediately* responsible, as indicated in the next section, but they are in the long run under the control of the genes. There is, however, a long interval between the egg and those characters, mostly in the adult, whose differences in different individuals reveal differences in the heredity units. What happens in this interval, to lead so faithfully to a given end result?

It is the ambition of geneticists and embryologists to bridge this gap. Part of it has been filled in, though sketchily. Most of the factors yet discovered concern the production of those major features, such as the nervous system, in which all individuals are sensibly alike, rather than in the minor features in which individuals differ. The principles involved must, however, belong to the same scheme; discovery of the agencies controlling production of general form and the organ systems is almost certainly progress toward an understanding of the smaller features of development. Only a few of the things already known can be pointed out.

Cytoplasmic Influence.—Cleavage of the egg follows a pattern that is frequently different in different species. The spindles of the dividing cells take certain positions in the cells so that the two cells resulting from the division have a fixed spatial relation to one another. It is possible, in such early blastulas, to say which cells were produced by one division, even when the division has not been observed. Lines of cell descent have been mapped in a number of such organisms.

When two species having different patterns of cleavage are crossed, the fertilized egg follows the pattern which it would have followed had it been fertilized by a sperm of the same species. That is, the type of cleavage is determined by the cytoplasm, practically all of which comes from the mother. This one-sided influence is lost before the hybrid begins to produce eggs, one generation later, for by that time the cytoplasm has been modified under the influence of the genes from both parents.

Symmetry.—Among higher animals the body usually possesses a bilateral symmetry, in that one half of it is a mirror reflection of the other. What determines the position of the plane between these halves? The egg often is spherical and has at the outset no observable symmetry to which the future body could be related. In some of the frogs, the plane of symmetry usually coincides with the first cleavage plane, that is, the plane between the first two cells derived from the egg. This plane passes through the two so-called poles of the egg, and in some frogs through the point of entrance of the spermatozoon which fertilizes it. In many other animals, however, there is no known relation of symmetry to cleavage.

In insects and their allies the egg itself often has a bilateral symmetry before cleavage, and the symmetry of the adult animal coincides with that of the egg. How early, and how, the symmetry of the egg is fixed is not known.

Organizers.—The exactitude of cell division, with its apparent duplication of the genes in the chromosomes, leads to the assumption that all the cells of an individual are genetically alike. Why, then, do some of them produce a nervous system, some a gill, some a heart, etc.? In general, it appears that the position of the cells in the whole embryo has an important influence on their fate. If their position is changed, their destiny may be altered. For example, if two patches of cells in the ectoderm of a salamander embryo, one from an area which would normally form part of the gills, the other from the place where a nervous system develops, are removed and each is inserted in the place of the other, the cells which would ordinarily become gill tissue become nervous system, and those which would naturally be nervous system become gill. Something about the place occupied by the cells helps decide what they shall be.

In the example used, the nervous system is caused to develop by the cells of the mesoderm beneath. If some of these mesoderm cells are taken out and inserted beneath the ectoderm at some place along the side of the body, an extra nervous system develops in the ectoderm over them. It has been shown that this influence of the mesoderm is exerted by some substance which the mesoderm cells contain. The mesoderm cells did not always possess this substance; they must have developed it, perhaps under the influence of some other cells, at an earlier stage. Thus there is a chain of events, the first links of which are still unknown. The substances, or other agencies if some of them are not substances, which exert such influences, have been called *organizers*. It is likely that much of embryonic development depends on them for guidance.

Time of Determination.—It is clear, from the account of organizers just given, that the fate of different parts of an individual is fixed at different times. There is perhaps not just one time at which an organ or tissue has its destiny settled, since in the chain of events leading up to its formation one agent may modify it at an early stage, another agent at a later stage. Nevertheless, taking into account the influences that are normally

exerted, some organs pass the stage at which modification is possible earlier than other organs pass that point. That is, some structures are "determined" earlier than others. In certain insects, for example, the reproductive system is determined early, the antennae late.

When the agencies that bear on development, be they external ones like temperature or internal ones like the organizers, change at a particular time of development, they are able to influence those structures whose nature has not yet been fixed, but they are too late to modify those parts which have been determined before that time. Thus the mere time at which something happens has an important consequence in directing the course of development. This time of occurrence of an event depends, in turn, on the rate of development of something leading up to it. Rates of development may be different at different stages, and the same agency (temperature, for example) may modify the rates of development of different organs unequally. This unequal effect on rates of development is no doubt one of the mechanisms by which environment influences embryogeny.

Autonomy in Development.—To what extent cells in an embryo or other immature individual develop in accordance with something which they themselves contain (their genes, for example) and how much they are influenced by other cells around them, or by body fluids whose nature may be determined by many parts of the organism, has been one of the fundamental problems of embryology. In vertebrate animals there is a great deal of mutual influence of part upon part, through the agency of chemical substances which are carried about by the blood or which diffuse from cell to cell. In insects, on the contrary, there is a considerable degree of autonomy, that is, cells or groups of cells become what the genes in them determine, though there are some exceptions.

This autonomy in the insects is illustrated by some very striking experiments of Beadle and Ephrussi (1936) in which they have transplanted rudiments of structures from one larva to another in the vinegar fly Drosophila. This fly has produced over a thousand mutations in the last 30 years and has been for this reason one of the principal objects of genetic study. When the rudiment of an eye in a larva which, when adult, would have a brown eye was transplanted into a larva that would develop

a wild-type red eye, the transplanted eye became brown, despite its surroundings. Twenty other mutant eye colors were similarly autonomous when transplanted. Only cinnabar eye and vermilion eye responded to the strange environment by becoming wild-type red; they were not autonomous. Wild-type red-eye rudiments, transplanted into mutant larvae, usually continued their normal development and became red; but when transplanted into prospective claret-eyed larvae, they became claret. Many other transplants were effected, and in a very great majority the transplanted rudiments were autonomous. The explanation of their various behavior is thought to lie in the production of a chain of three substances, the second and third of which depend on the prior presence of the first.

Genetic Identity of All Cells in Individual.—Although as suggested earlier the manner in which cells divide leads one to conclude that all cells in one individual contain identical genes, and most biologists have so concluded, there remains a possibility that some mutation of genes occurs as a part of embryonic development. One competent geneticist has pointed out a situation in which such developmental mutation would provide a simple explanation of observed phenomena. Wright (1934) suggests that certain spotted color patterns in mammals are due to the change or mutation of pigment genes at definite places in the body, after a regular scheme and in response to some physiological gradient.

Gradients are known in development, a very widespread one being the anteroposterior graduation of time or rate of development. In many animals development starts earlier at the anterior end than at levels farther back. The embryo of a vertebrate animal produces its first muscle segments well forward, while new segments are added successively behind them. A crayfish embryo develops its front appendages earlier than its posterior ones; and, at any given stage until their development is complete, the front ones are more advanced than the posterior ones. Some other gradients are known, and they are probably quite common. Even if such gradients do not induce mutation, they may exert an influence of a nongenetic sort on development, which would be on a par with organizers in general.

Convergence of Genetics and Embryology.—The preceding sections probably contain as much as can profitably be said

concerning developmental determination in advance of a full presentation of the mechanism of heredity. The examples used will serve to illustrate the types of problems with which the embryologist is confronted. It should be evident that the experimental embryologist, with transplantation as a tool, and the geneticist, using now the same method but with a more minutely analyzed background of comparison, are converging upon the answer to the same question: What makes organisms what they are?

CHAPTER VI

MECHANISM OF HEREDITY

From the preceding chapters it will have been correctly inferred that heredity is governed by a considerable number of minute bodies, the *genes*. These bodies are probably protein substances, and may be either single molecules or small groups of them. They are contained in the chromosomes, being placed in a single row from end to end of these structures. Though two or more identical genes may occur in the same chromosome, the genes of one chromosome are mostly different from one another. Probably they differ greatly in size, but the important differences among them must be chemical.

Identity of the Genes.—Most of the things just said concerning genes were earlier said of the nodules or chromomeres of which the chromosomes are in part composed. To what extent the genes may be identified with the chromomeres is uncertain. Some cytologists, working with cells in which the chromomeres are small and very numerous, have not hesitated to assume that the visible knots are the genes. In most cells, however, there are not enough of the little pellets to permit this assumption. In the vinegar fly Drosophila, which has furnished more of our knowledge of heredity than any other organism, calculations of several sorts indicate the total number of genes to be from 1800 to 14,000 in a mature germ cell. Probably other organisms of similar complexity have comparable numbers of them. In very few animals or plants, however, can anything like even 1800 chromomeres be detected in the chromosomes of a mature egg or spermatozoon. Whether the chromomeres are aggregates of genes or whether only a few of the genes are visible and hosts of others are beyond the limits of visibility cannot now be stated.

In one tissue of Drosophila, however, the chromosomes are greatly enlarged, and in these it is possible that the genes are actually being seen. That tissue is the salivary gland. It had long been known that the chromatin of the salivary gland cells of flies is in the form of heavy cords marked by crossbands. These

ropelike strands were in appearance like great rolls of pennies. Partially spread out, they are shown in Fig. 29. It was hoped that the disks might give some clue to the organization and individuality of the chromatin, and Painter (1933, 1934) found this to be true to a very remarkable degree. With a little stretching the salivary gland chromosomes are from 100 to 150 times as long as the corresponding chromosomes in germ cells. They are likewise thicker, though this dimension is much less exaggerated. Some of the thickness appears to be due to a multiplication of the chromosomes, for each so-called chromosome

FIG. 29.—Salivary gland chromosomes of Drosophila. Entire group of chromosomes, attached to geneless granular material in the center. (*Modified from Painter, Journal of Heredity.*)

shows signs of being composed of eight to sixteen strands all closely joined to one another.

The disks which give the appearance of crossbands are very different in size and shape (Fig. 30). Some are heavy, some light, with all sizes between; some appear to be continuous unbroken disks, others are divided into separate segments; some are solid, others hollow like very thin flasks. These identifiable disks are distributed along the chromosome according to a very definite pattern, and this pattern is the same, for the corresponding chromosomes, in every cell of the gland. It is accordingly possible, when only a part of a chromosome is seen, to say to which chromosome it belongs and where in the length of that chromosome it is placed.

The differences between these disks, and the definite pattern of their arrangement, are suggestive of the differences among genes and the pattern of their arrangement in chromosomes, as

discovered not by observation but from breeding experiments. The correspondence between disks and genes in these two respects raised the question whether the disks may not be the genes. The answer to that question is now being sought. By X rays or by accident, the chromosomes can be broken and parts misplaced or omitted, and it is found that the mode of inheritance of certain characters is at the same time changed. By noting what characters are thus modified, and what bands of the chromosomes

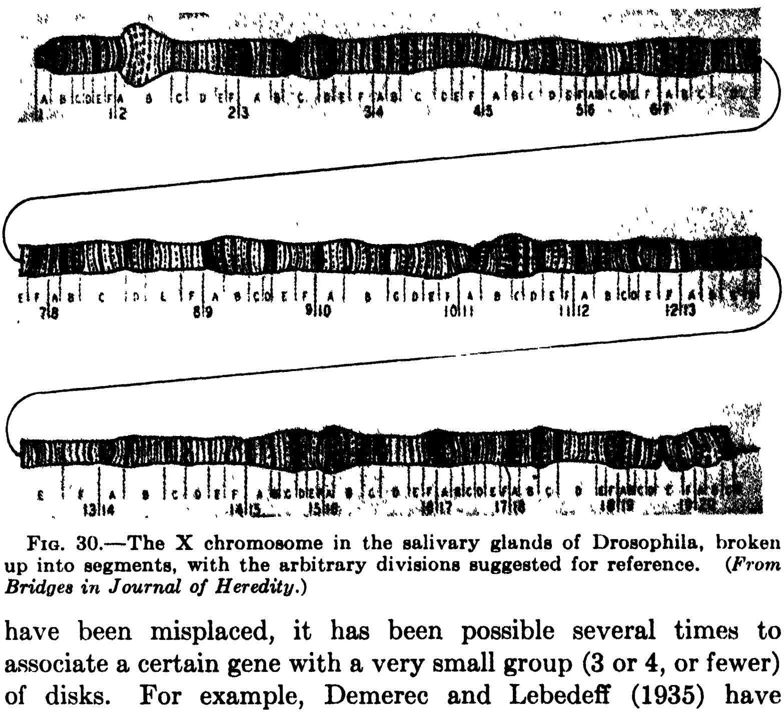


FIG. 30.—The X chromosome in the salivary glands of Drosophila, broken up into segments, with the arbitrary divisions suggested for reference. (*From Bridges in Journal of Heredity.*)

have been misplaced, it has been possible several times to associate a certain gene with a very small group (3 or 4, or fewer) of disks. For example, Demerec and Lebedeff (1935) have studied stocks of flies in which the character "forked bristle" is modified and have found that in each of them the disk designated 15F1 in the map (Fig. 30) by Bridges (1935) is among those missing. Nothing as yet forbids the assumption that the visible disks are, or contain, the genes, and that assumption may well be provisionally made.

Genes in Homologous Chromosomes.—As has already been stated (page 30), the chromosomes of a cell are of two similar sets. Each chromosome is matched by another one very much like it, that is, the chromosomes are twins. One chromo-

some of each pair came from the mother, the other from the father.

The similarity of these chromosomes extends to their genes. Similar genes, arranged in identical pattern, mark the homologous (page 31) chromosomes. This similarity can readily be seen in the salivary gland of Drosophila. Unlike those of most cells, the similar chromosomes of this gland are united side by side. There *appears* to be in each cell only one chromosome of a kind, instead of two, but that is only because the like chromosomes are joined. In this uniting, the corresponding bands (genes?), being placed in the same order and the same distance apart, lie opposite one another. Now, so great is the similarity of the disks in the two chromosomes that each pair of disks makes a single unbroken band crossing both chromosomes. Only

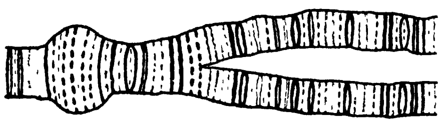


FIG. 31.—Correspondence of gene pattern in homologous chromosomes. Portion of so-called second chromosomes of Drosophila, joined together at left, separate at right. The two patterns are identical.

occasionally do the chromosomes fail to join firmly at some places in their length (Fig. 31), but at these places the similarity of the bands at the same level is still plainly visible.

In other types of cells, it can often only be inferred that similar genes occupy the corresponding loci of the homologous chromosomes. In Fig. 9 there are similar chromomeres at the same levels, but what relation these nodules bear to genes is uncertain. Nevertheless, there is a great body of evidence of an experimental sort, some of which is presented in later chapters, from which the location of genes may be ascertained. The order of the genes and their distances from one another may thus be learned, that is, maps of the chromosomes can be made, and hundreds of genes have been assigned their proper places in the chromosomes of Drosophila. Now, since the two homologous chromosomes of any pair can be traced separately in different lines of descent, it is possible to show that the chromosome maps of both the homologues are identical. And so, even if the genes cannot be observed, it may still be known that two twin chromosomes have similar genes placed at the same levels in their length, and

that if these chromosomes were placed side by side as in the salivary glands similar genes would be opposite one another.

Early Stages of Maturation in Animals.—Just such an apposition of the chromosomes side by side actually occurs in the germ cells of animals as they start the long process known as *maturation* or *meiosis*. Prior to the beginning of meiosis, the reproductive cells, *spermatogonia* in the male, *oögonia* in the female, multiply by repeated cell divisions of the ordinary duplicating type. When the animal reaches a given stage, some of these cells cease to divide by ordinary division. These cells are now *primary spermatocytes* and *primary oöcytes*, in the respective sexes. It is in these cells that the pairing of the chromosomes referred to above takes place. The homologous chromosomes, maternal and paternal, come together side by side in pairs. Though the

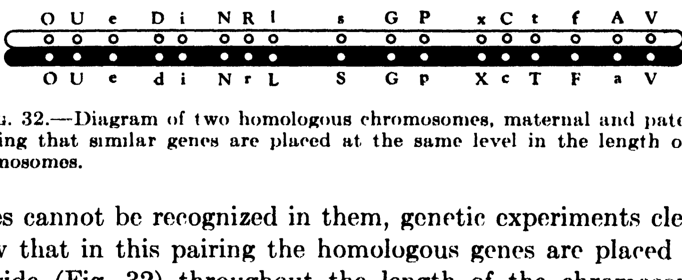


FIG. 32.—Diagram of two homologous chromosomes, maternal and paternal, showing that similar genes are placed at the same level in the length of the chromosomes.

genes cannot be recognized in them, genetic experiments clearly show that in this pairing the homologous genes are placed side by side (Fig. 32) throughout the length of the chromosomes. It is practically certain that the similar genes have some attraction for each other and that it is the pairing of the genes which causes the pairing of the chromosomes.

While the chromosomes are pairing, the cells containing them grow in size—the primary spermatocytes moderately, the primary oöcytes enormously as a rule. The upper four rows of cells in Fig. 33 represent, first, the multiplication of the cells, and then their growth and the pairing of the chromosomes. In so condensed an illustration the paired genes cannot be shown.

The Divisions in Meiosis.—The rest of maturation consists largely of two successive cell divisions, one following close upon the other. In one of these divisions the chromosomes are duplicated as in ordinary cell divisions; in the other division they are not duplicated, but some go to one cell, some to the other. The division in which the chromosomes are merely separated into two groups is known as the *reduction* division because the

number of chromosomes per cell is thereby reduced to half. The division in which the chromosomes are duplicated is called the *equation* division, referring to the equality of the cells produced. Ordinary cell division elsewhere is likewise equational.

Which of these types of division occurs first is immaterial in most respects, though many animals follow a regular scheme. In some species reduction takes place in the first division; in others the second division is reductional; in still others some of the chromosomes experience reduction in the first, the other chromosomes in the second division; and finally, in some organisms corresponding chromosomes behave differently in different germ cells of the same individual.

Reduction Division.—The diagram in Fig. 33 represents reduction as occurring first. The homologous chromosomes, having previously paired, now part company, one going to one cell, the other to the other cell. It will be appreciated that in this separation, the homologous genes are likewise separated. Just as the daughter cells get only one chromosome of each pair, so do they get only one gene of each pair. The total number of chromosomes in each daughter cell is just half as large as the number in the cell from which they are derived. This reduced number is called the *haploid* number, as contrasted with the original or *diploid* number.

The several pairs of chromosomes are independent of one another in this separation, that is, the paternal member of one pair may go to the right, but that of another pair to the left. To which of the two cells a given chromosome goes is mostly a random matter. Consequently, various combinations of maternal and paternal chromosomes are found in the different cells resulting from the reduction division. In some such cells all the chromosomes are paternal, in some all are maternal, and in others there are all conceivable combinations of maternal and paternal. Since each chromosome contains certain genes, the germ cells therefore contain all sorts of combinations of genes.

In the male (Fig. 33, left), the first meiotic division produces two equal cells, both functional, which are known as *secondary spermatocytes*. In the female the division is very unequal, one daughter cell being very large (*secondary oöcyte*), the other very small (*polar body*). Only the large cell in the female is functional; the polar body gradually disintegrates and disappears.

Equation Division.—Each cell produced by the first maturation division, with the exception usually of the polar body, immediately prepares to divide again. If the first division was

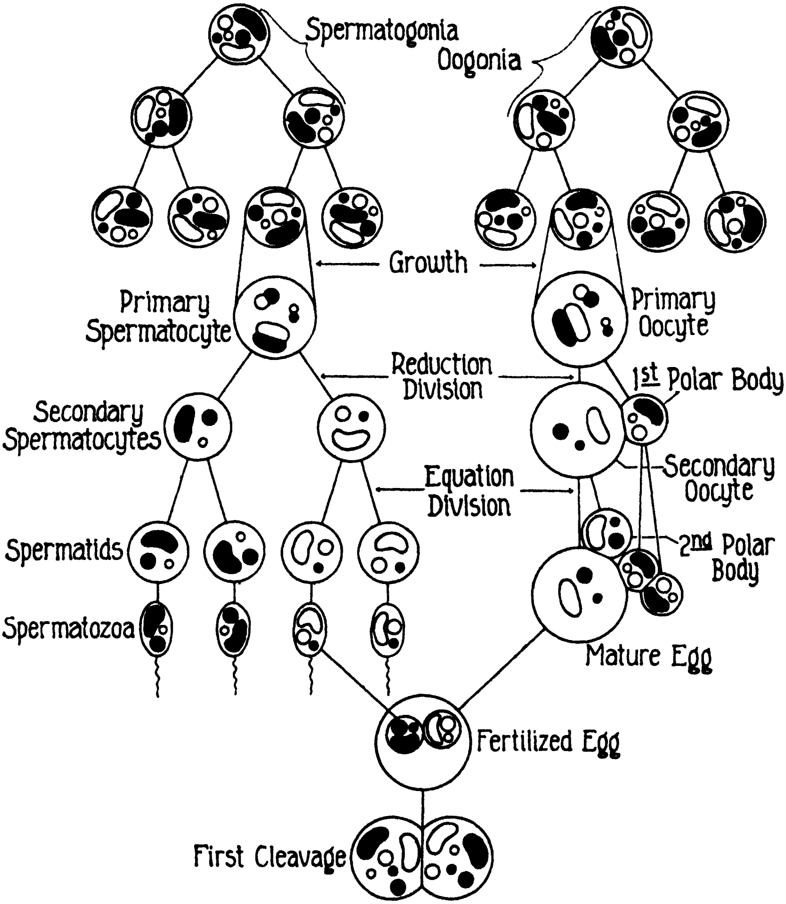


FIG. 33.—Diagram of maturation or meiosis of germ cells in animals. The process begins with the third row of cells. Maternal chromosomes white, paternal black. All chromosomes in the fertilized egg received from the mature egg are thereafter maternal, and those received from the spermatozoon are thereafter paternal, regardless of what they were in the mature germ cells.

reductional for any cell or any pair of chromosomes, this one is equational. The chromosomes are duplicated, and two cells genetically identical with one another and with the parent cell are produced by each division.

These cells in the male are equal, and are called *spermatids.* From one primary spermatocyte come four spermatids. If the reduction division occurred first, two of the spermatids are identical and the other two identical but different from the first two. In the female the division is again very unequal, the large cell being the *mature egg*, the small one another polar body. To distinguish the polar bodies produced by the two divisions, they are called *first* and *second*, respectively. In a few animals, the first polar body, though nonfunctional, divides equationally as does the secondary oöcyte. This kind of animal, though unusual, is represented in Fig. 33 in order to complete the comparison of meiosis in the female with that in the male. From one primary oöcyte are derived, in such an animal, four cells; but only one of the four, the egg, ever functions further.

The spermatids change shape to produce the mature *spermatozoa*, and maturation is finished.

Summary of Meiosis.—The features of meiosis which are of greatest significance in heredity are the following: (1) the pairing of the homologous maternal and paternal chromosomes; (2) the separation of these paired chromosomes and their passage to different cells in the reduction division; (3) the consequent separation of the genes of each pair to different germ cells; (4) the independence of the several pairs of chromosomes in this separation; (5) the resultant assembling of various combinations of maternal and paternal chromosomes in the different mature germ cells; (6) the variety of combinations of genes thus produced in the different germ cells; and (7) the reduction of the number of chromosomes in the mature germ cells to half that found in the reproductive cells before maturation.

Each of these features has important consequences to which attention must be called in later chapters. It is essential that their source in the mechanism described in the preceding section be thoroughly understood.

Fertilization.—In most animals employing sexual reproduction, an egg must be fertilized before it can develop into a new individual. In this process a spermatozoon enters the egg either after the maturation is completed or at some earlier time during the maturation process. Eventually the nucleus of the spermatozoon approaches that of the egg until they are side by side. As the fertilized egg prepares for cleavage, the membranes of

the two nuclei dissolve away, leaving the chromosomes of both parents free to enter the spindle of the dividing cell.

The designations maternal and paternal as applied to the chromosomes frequently change at this point. When an egg is fertilized a new individual is started. The chromosomes in the fertilized egg must therefore be labeled with reference to the parents which contributed to it. In Fig. 33, though the egg is represented as containing one maternal and two paternal chromosomes, these are all maternal in the fertilized egg and are so represented in the illustration. The chromosomes in the spermatozoon are two maternal and one paternal; but in the fertilized egg they are all paternal. The descendants of these chromosomes retain their new designations throughout the new individual, including its mature germ cells; but, in the long run, half of the chromosomes will change names again when these germ cells participate in fertilization and the formation of new individuals in the next generation.

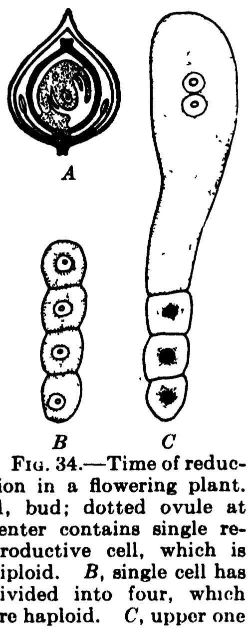


FIG. 34.—Time of reduction in a flowering plant. *A*, bud; dotted ovule at center contains single reproductive cell, which is diploid. *B*, single cell has divided into four, which are haploid. *C*, upper one of these four growing and dividing to produce embryo sac, other three degenerating. (*A from Sinnott, Botany: Principles and Problems, McGraw-Hill Book Company, Inc.*)

Reduction in Flowering Plants.—The change from the diploid to the haploid chromosome number, by the retention of only one chromosome from each pair of homologues, occurs somewhat earlier in flowering plants than in animals. In the bud shown in Fig. 34, the dotted portion in the middle consists of the ovule, including the future seed coats. In the midst of the ovule is a single cell from which the egg is later derived. This cell is diploid; but, when it is twice divided to form a row of four cells (Fig. 34, *B*), each of these cells is haploid. Reduction has taken place in one of the two divisions, and the available evidence indicates that it is usually the first division.

Three of these four haploid cells degenerate (Fig. 34, *C*), while the fourth divides three times to form the eight cells of the embryo sac (Fig. 16, *f.g.*). All of these eight cells are

haploid. One of them is the egg, as was earlier indicated (page 39). There is thus a series of three or four cell divisions following reduction before the egg is finally formed, as compared with only one division, or none at all, after reduction in animals.

Reduction is similarly early for the male cells of plants. The cells of the last diploid generation in the anthers divide

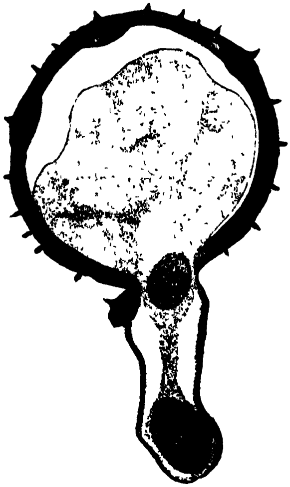


FIG. 35.

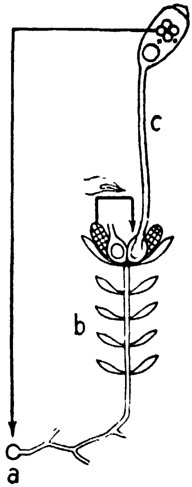


FIG. 36

FIG. 35.—Pollen grain, germinating. The pollen tube nucleus is at the tip of the growing tube. Behind it is the generative nucleus, which later divides into two. (*From Sinnott, Botany: Principles and Problems, McGraw-Hill Book Company, Inc.*)

FIG. 36.—Diagram of life cycle of a moss. *a*, spore, *b*, gametophyte, or moss plant; *c*, sporophyte. The spore and gametophyte have half as many chromosomes per cell as does the sporophyte. (*After Bělař in Handbuch der Vererbungswissenschaft.*)

into four cells which are haploid. Reduction appears to occur usually in the first of the two divisions which produce these four cells. Each of the four haploid cells then proceeds to form a pollen grain, by dividing into two cells, both haploid, only one of which has a reproductive function. As the pollen grain germinates and grows down the style of the female parts of a flower (Fig. 16), the reproductive nucleus, which lags behind the nonreproductive one (Fig. 35), divides into two. One of these two fertilizes the egg, the other unites with two nuclei near the egg to form the endosperm nucleus, as explained before (page 40). In the male cells, therefore, there are two or three divi-

sions between reduction and the final completion of the reproductive nuclei.

Reduction in Mosses.—In some of the lower plants, such as ferns, mosses, and liverworts, there is a much longer interval between reduction and the production of germ cells. The moss cycle will serve to illustrate. A moss spore (Fig. 36, *a*), falling to the moist soil, germinates to produce a branching chain of cells. From some of these cells a moss plant, called the *gametophyte* (*b*), develops. At the top of the gametophyte the germ cells, male and female, are produced. All the cells so far mentioned, from spore to germ cells, are haploid.

The egg is then fertilized, and from the fertilized egg develops a club-shaped structure, called the *sporophyte* (*c*). This structure is diploid, as was the fertilized egg from which it came. But when the sporophyte produces spores, to repeat the cycle, these spores are haploid again. One of the last two divisions by which the spores are produced is a reduction division. Not until the moss plant (gametophyte) is mature, however, are germ cells again produced. In the mosses, therefore, many thousands of haploid cells are produced after reduction and before the germ cells are formed.

Reductional Nondisjunction.—As an exceptional occurrence, separation of a maternal chromosome from its paternal homologue may not take place. Both chromosomes then go to one cell, while the other cell receives no chromosome of that pair. This nondisjunction is comparable to that of two duplicate chromosomes in ordinary cell division (page 32); but since maternal and paternal chromosomes may not be exactly alike, the consequences of reductional nondisjunction are somewhat different.

CHAPTER VII

SIMPLEST PHENOMENA OF HEREDITY

The relation of genes to chromosomes was gradually discovered while knowledge of the chromosomes was accumulating. It was only a few years after chromosomes were first observed that a theory connecting them with the processes of heredity was promulgated. From that time on, knowledge of chromosomes and understanding of heredity advanced together. Sometimes a new discovery regarding chromosomes suggested a new feature of inheritance which was later confirmed. More often some result of a breeding experiment, demonstrating a new relation in heredity, indicated a novel aspect of chromosomes which was then found to be real. The latter order has been the common one with respect to the finer details of the genetic mechanism. Most of what is known regarding the order of genes in the chromosomes, their distances apart, and the accurate side-by-side pairing of the genes as the chromosomes pair in meiosis was merely inferred from the manner in which characters were inherited in crosses. Such observational confirmation of these details of architecture as is found in the salivary gland chromosomes of Drosophila came very late—long after the general scheme of gene arrangement had been thoroughly established.

Hybridization the Source of Knowledge of Heredity.—In the preceding chapter the mechanism of heredity was described in terms which may have seemed to imply that the details of chromosome structure could be seen. It is only in such remarkable cells as those of the salivary glands of flies, however, that many details are actually visible; and even in these giant chromosomes the real meaning of the elements must be determined from crosses between different kinds of individuals. While the quickest and easiest way to master the principles of heredity, if one does not have to make his own discoveries, is to start with the chromosomes and genes, it must not be forgotten that advancement of knowledge regarding fine details was

attained by the opposite procedure. Crosses were effected between individuals differing in some character or characters, the offspring in one or more succeeding generations were found to display certain qualities or combinations of qualities in certain numbers of individuals, and then a logical scheme was devised to account for the observed results. When large numbers of crosses led to the same scheme, differing in details which were comprehensible but agreeing in the fundamental plan, that scheme stood demonstrated as the mechanism of heredity.

Simplest Monohybrids.—The simplest sort of experiment to explain by a logical scheme is one in which two organisms differing in just one respect are crossed and in which the genes contained in any individual can be known, once the scheme is understood, from a mere visual inspection of that individual. The old-fashioned garden flower known as the snapdragon furnishes a number of examples of this simple sort. One of them involves the red, ivory, and pink colors of the flowers.

A red-flowered snapdragon breeds true, that is, if it is self-fertilized, its offspring are all red-flowered. Ivory-flowered plants likewise breed true. Now, when these two types are crossed, by pollinating either a red flower from an ivory one or an ivory flower from a red one, seeds are produced which develop into offspring with pink flowers (Fig. 37). The color is intermediate between the colors of the parents, and all the offspring are alike in this respect. These pink plants are known as the F_1 generation, abbreviated from *first filial.*

Now, if a pink-flowered plant of the F_1 generation is self-fertilized or if two pink ones are crossed, the seeds thus produced give rise to an F_2 (*second filial*) generation which consists of three kinds of plants. About $\frac{1}{4}$ of the F_2 plants have red flowers, about $\frac{1}{2}$ of them are pink, and $\frac{1}{4}$ are ivory (Fig. 37).

By self-fertilizing any of the F_2 plants an F_3 generation is obtained; but the nature of the F_3 plants depends on which of the three types of F_2 plants was self-fertilized to produce them. A red-flowered plant yields F_3 that are all red; an ivory F_2 produces only ivory; while a pink F_2 gives three kinds of offspring in F_3, $\frac{1}{4}$ red, $\frac{1}{2}$ pink, $\frac{1}{4}$ ivory.

An F_3 generation can also be obtained by crossing different kinds of F_2 plants. However, some of the results are identical with those already briefly stated, and one of them involves a

different type of mating, which is described in a later chapter. These other sources of F_3 are therefore not considered here.

Explanation of Color Inheritance in Snapdragons.—The very regular numerical results just described have a simple explanation in the random distribution and recombination of the

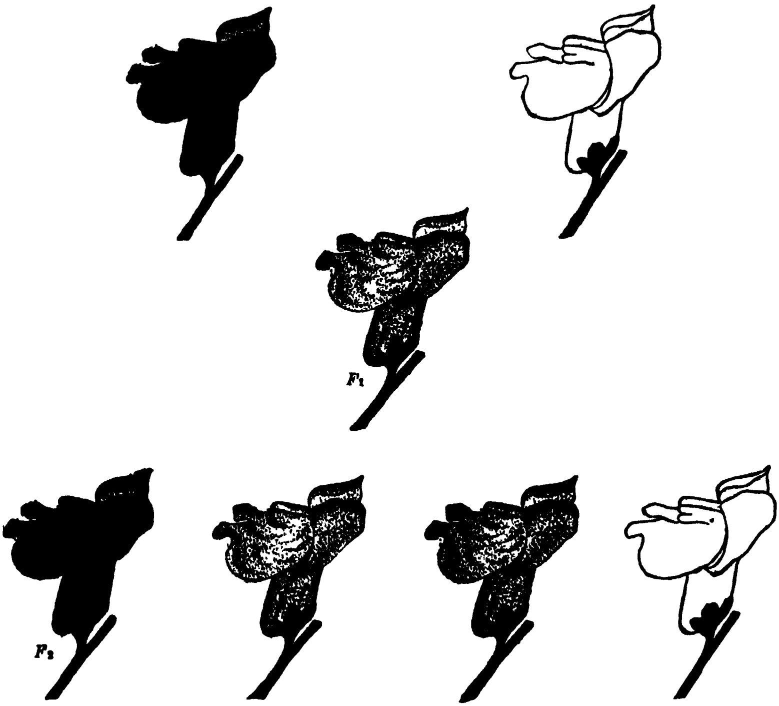


FIG. 37.—Cross between a red snapdragon (represented dark) and an ivory snapdragon (shown white). The offspring (F_1) are intermediate in color, that is, pink. The F_2 generation produced by self-fertilizing a pink F_1 flower consists of red, pink and ivory flowers in the ratio indicated by the illustrations.

chromosomes and genes. The red and the ivory plants differ with respect to the genes of only one pair; all other pairs of genes are alike in the two plants. These genes, it must be assumed, are in the homologous pairs of chromosomes in the two plants (Fig. 38). The differentiating genes in the ivory plant may be symbolized by the letter i, those in the red plant by I.

At some time prior to the formation of the germ cells, as explained in the preceding chapter, the reduction division

separates the chromosomes of the pair that contains *I* or *i*, so that each cell has only one of these chromosomes. The eggs of the red-flowered plant all contain the gene *I*, the pollen grains of the ivory plant all contain the gene *i*. When the egg is fertilized by one of the generative nuclei of the pollen, the seed containing the fertilized egg produces a plant having one chromosome with *I* and the other chromosome of this pair with *i*. The combined action of *I* and *i*, along with the many other genes in the plant, causes the flowers to be pink.

When the F_1 pink-flowered plants produce their germ cells, two kinds of eggs and two kinds of pollen are formed. Sometimes the reduction division in the female cells occurs in such a way that the surviving upper one of the four cells (Fig. 34, *C*) contains the maternal chromosome and its gene *I*, and from this cell is developed the embryo sac, including an egg. In other instances the reduction division carries the paternal chromosome and its gene *i* to the surviving upper cell, which then proceeds to produce an embryo sac containing an egg. Since the reduction division is just as likely to carry the one chromosome as the other to this surviving upper cell, the two kinds of eggs *I* and *i* should be about equally numerous.

The male reproductive cells in the anthers behave in essentially the same way as do the female cells, except that all cells survive. After the reduction division, there is one cell containing the maternal chromosome with *I*, another cell containing the paternal chromosome with *i*. The descendants of these cells produce pollen, which therefore is of two kinds *I* and *i*, each kind exactly as abundant as the other. Figure 38 shows the two kinds of eggs and two kinds of pollen.

Both kinds of pollen fall on the stigma, and their tubes grow down through the style and reach the two kinds of eggs at random. Four combinations are possible, and, since they are effected at random, they should be equally numerous. These four combinations *II*, *Ii*, *iI*, and *ii* are shown at the bottom of Fig. 38. They produce red, pink and ivory plants. Since the combinations *Ii* and *iI* are in effect identical, the pink-flowered plants are twice as numerous as either the red or the ivory.

From the diagram it is likewise clear why the F_3 generation differs in different families, depending on which type of F_2 plant produces it. Red can produce only red if self-fertilized,

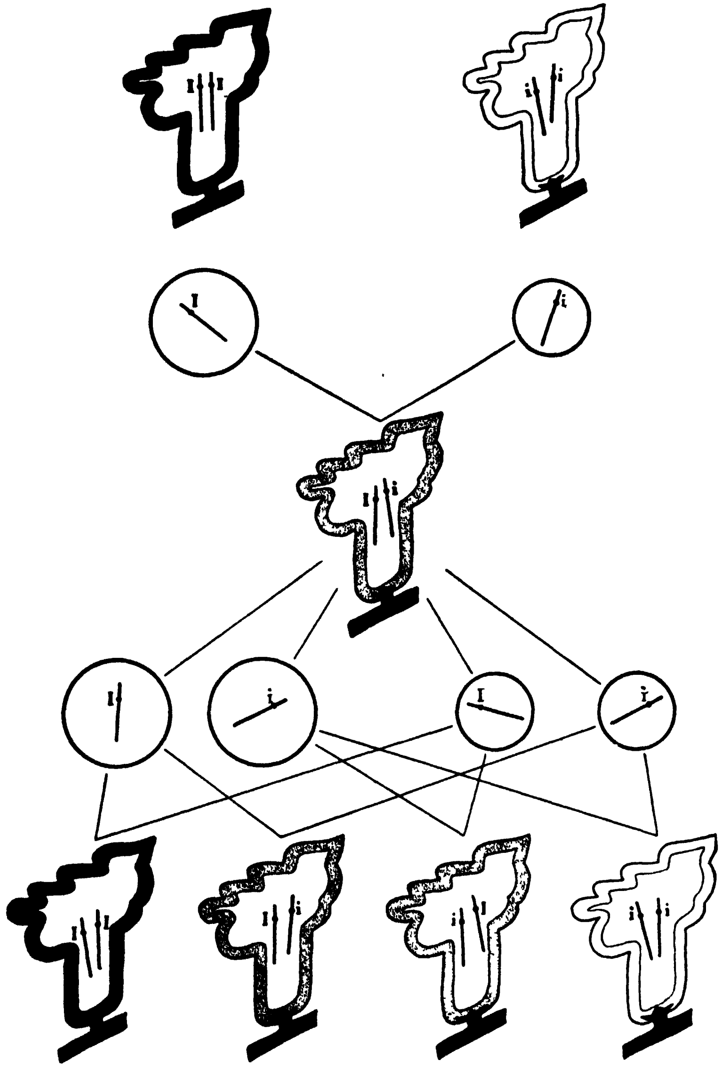


FIG. 38.—Diagram representing inheritance of color in snapdragons. Black border, red flowers; dotted border, pink; white border, ivory. Large circles, eggs; smaller circles, pollen cells. Heavy lines, chromosomes; dots in the chromosomes, genes. *I*, gene for red; *i*, gene for ivory.

because all eggs and pollen must contain I. Ivory plants, self-fertilized, can produce only ivory, since all germ cells will contain i. Pink flowers, self-fertilized, will, however, yield the three kinds of offspring, in the same proportion as in the F_2 generation.

Terminology.—To refer to the several kinds of individuals involved in the crosses described, with respect to their combinations of genes, certain terms are useful. Any organism in which the two genes of a given pair are alike (II or ii) is called a *homozygote*. One whose genes of any pair are different (Ii) is said to be a *heterozygote*. The terms come from the word *zygote*, which means the combination produced by the union of two cells to form one cell in reproduction. A fertilized egg is thus a zygote. By an extension of meaning, individual animals or plants which have developed from fertilized eggs are also called zygotes, when it is desired to distinguishing them from their germ cells, which are called *gametes*. Zygotes are diploid with respect to their chromosomes and have two genes of each pair, gametes are haploid and have but one gene of each pair.

Two other useful words relate to the distinction between the genes an organism possesses and its visible appearance or other qualities. The aggregate of genes in an animal or plant, or the group of genes under consideration, is called its genetic composition, or *genotype*, sometimes simply its heredity formula. Its observable qualities are, by contrast, called its *phenotype*. The phenotype of the F_1 snapdragons in the foregoing experiment is pink; their genotype is Ii.

Distinctive Feature of Simplest Heredity.—The simplest operations of heredity must concern only one pair of differentiating genes. The additional feature of the snapdragon contrasts which makes them particularly simple is that there is only one genotype for each phenotype. The pink-flowered plants are always of the genotype Ii, the red ones always II, and the ivory ones always ii. The genotype may be recognized by merely observing the quality of the individual. In many characters this is not true, as will be seen in the next chapter, for organisms of the same phenotype often have different genotypes.

Other Examples.—A considerable number of other plants, and of animals, show this unequivocal correspondence between genotype and phenotype. In the weed shepherd's-purse one variety has in the young or rosette stage a leaf whose main

expanse is at the tip (Fig. 39, left). In another variety the blade of the leaf is broadly lobed (right). The hybrid produced when they are crossed has a leaf with irregular but rather narrow lobes (center). The leaves in various positions on the plant have different forms, but nearly every one in the heterozygote is unlike the corresponding leaf of either homozygote.

Roan color in Shorthorn cattle (Fig. 40) is an equally good example. This pattern consists of hairs of two colors, white

FIG. 39.—Distinguishable heterozygote in shepherd's-purse Rosette leaves of parents left and right, their hybrid between. (*Photograph by Professor G. H. Shull.*)

and either red or black, irregularly interspersed among one another. There may be patches of considerable size which are mostly red, other areas mostly white; but often there are only a few hairs of the same color together. The roan pattern appears only in heterozygotes. White Shorthorns are homozygous (*ww*), red Shorthorns are likewise homozygous (*WW*). Their hybrid (*Ww*) is roan.

Distinguishable Heterozygotes in Man.—In man, while it is probably rather common for heterozygotes to be different from both homozygotes, not many simple examples are available. So many human characters are noticeably affected by several pairs of genes that the differentiating effect of a single pair is obscured. In the absence of controlled breeding experiments,

such as are possible with other animals and with plants, it is difficult to isolate the consequences of a single pair of genes. Yet there are many indications that in the interactions of several pairs the effect of *Aa* is often different from that of either *AA* or *aa*.

Not thoroughly established as a simple example of distinguishable heterozygote is the condition known as brachyphalangy, in which one segment of each finger or toe is reduced, that is, in

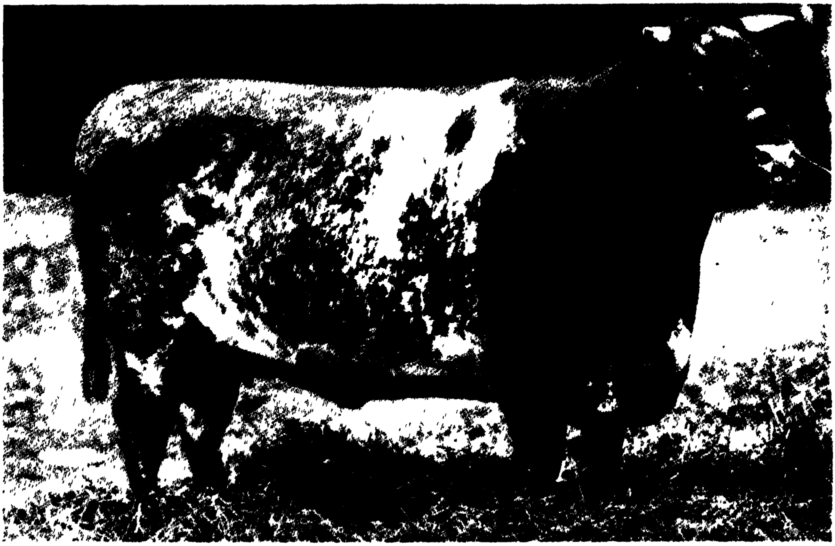


FIG. 40.—A roan Shorthorn cow. The coat consists of red hairs and white hairs interspersed, a condition found only in animals heterozygous for red and white. There are also blue roans, which are heterozygous for black and white. (*From McPhee and Wright, in Journal of Heredity.*)

heterozygotes. Very few people homozygous for this character are known, but the consequence of homozygosis seems to be considerable abnormality of the skeleton as a whole. This character is discussed again among lethal characters in Chap. XII.

A possible additional example of a human character in which each genotype is recognizable from its phenotype is the absence of the lateral incisor teeth in a family described by Keeler and Short (1934). This character recurred in each generation, presumably mostly in heterozygotes, since usually only one parent of a family lacked the incisors. When, however, both parents lacked the two lateral teeth, their child lacked five teeth. This child could have been a homozygote, and the fact that the

character was more marked in it suggests that being homozygous accentuated the abnormality. If homozygotes regularly have this heightened expression of the character, as compared with heterozygotes, it is again possible to recognize the genotype from the phenotype.

Certain substances in human blood are inherited in a way which, while different from that described in this chapter, nevertheless enables one to distinguish a heterozygote from both corresponding homozygotes. The substances are known as *agglutinogens* and are located in the red cells of the blood. Two of these substances are alternative to one another in the sense that only one of them can be represented in any gamete. They are named A and B. A person homozygous for A develops only A, and one homozygous for B has only B; but a person heterozygous for the two possesses both of them. The presence of either or both can be detected by laboratory tests. There is also another pair of agglutinogens named M and N, and their mode of inheritance is identical with that of A and B, in that one homozygote possesses M, the other homozygote has N, while the heterozygote has both M and N. In neither of these pairs of agglutinogens is the heterozygote intermediate between the two homozygotes, as pink snapdragons are; it has rather a combination of the qualities of the homozygotes, and so is readily recognizable. These agglutinogens are part of a somewhat more complicated system which is described further in Chap. XI.

CHAPTER VIII

DOMINANCE

When each phenotype can have only one genotype, breeding experiments are simple. If it is desired to cross two individuals possessing certain genes, such individuals can always be selected merely from their appearance. It is not necessary to know their parentage; an individual of unknown source will give quite as predictable results as one whose ancestry is on record. These statements are predicated upon the assumption that the mode of inheritance of the character has already been ascertained and that it is of the type exhibited by the snapdragons, shepherd's-purse, and cattle of the preceding chapter.

Many characters, however, do not reveal the genotype of the animal or plant exhibiting them. Some individuals may be either heterozygotes or homozygotes, and, to discover which one they are, it is necessary to obtain offspring from them, or to investigate their parentage.

Black and Brown Mice.—A familiar example of this uncertainty of the genotype is furnished by the black color of mice, as contrasted with brown coat. Black mice may be homozygous, or they may be heterozygous for brown and black. If a stock of mice has been inbred for some time and only black animals have been produced, these mice are assuredly homozygous. But a black mouse obtained from an unknown source may be heterozygous. Brown mice, on the contrary, are always homozygous for brown.

These relations of brown and black are ascertained from a cross like that shown in Fig. 41. If a black mouse, known to be homozygous from long inbreeding of its ancestors, is crossed with a brown mouse, sure to be homozygous without knowledge of its ancestry, all their offspring (F_1 generation) are black and indistinguishable from the black parent. This result is described by saying that black coat is *dominant*, brown *recessive*. When these black F_1 animals are interbred, the F_2 generation obtained from them consists of some black and some brown animals, the blacks being about three times as numerous as the browns.

Inheritance of Color in Mice.—The two kinds of mice differ only in one pair of genes, which are located in a pair of homologous chromosomes. The differentiating genes of the brown animals may be symbolized by the letter *b*, those of the black mice by *B*. The original black parent in this cross had two chromosomes containing *B* in each cell; but the reduction division left only one of these chromosomes, hence only one gene *B*, in each of its mature eggs (Fig. 42). The brown mouse had two chromosomes containing *b*, but the reduction division resulted in spermatozoa having only one chromosome of this pair and only one gene *b*.

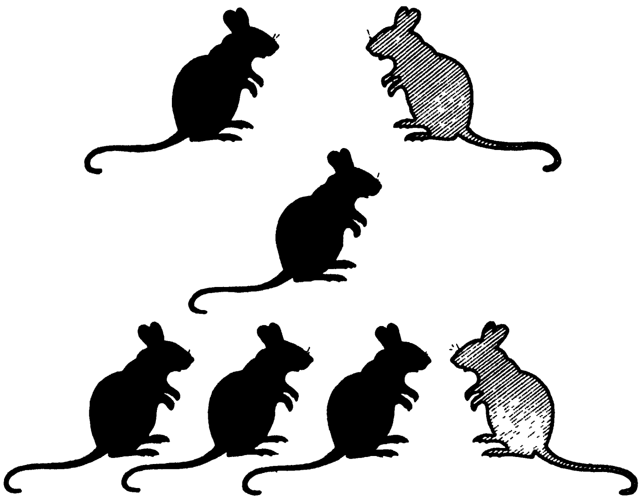


FIG. 41.—Inheritance of color in mice. Black figures, black mice, shaded figures, brown. The F_1 generation is black, the F_2 black and brown in the ratio of 3:1.

The fertilized eggs must therefore have had one chromosome with *B*, the other with *b*. These fertilized eggs developed into black mice. Why *Bb*, along with the other genes of the animal, should produce as black a mouse as *BB* does is unknown. For some reason a single *B*, even in the presence of *b*, produces as much black pigment (at least so far as the eye can see) as do two *B*'s. This capacity of *B*, even when *b* is present, to produce as great a visible effect as *BB* is what constitutes its dominance. If *I* in snapdragons had this capacity, the F_1 plants would have red flowers instead of pink.

The black F_1 mice produce two kinds of germ cells. In the reduction division in a female the chromosome containing *b*

may pass to the polar body, yielding an egg with *B*; or the chromosome with *B* may go to the polar body, producing an egg

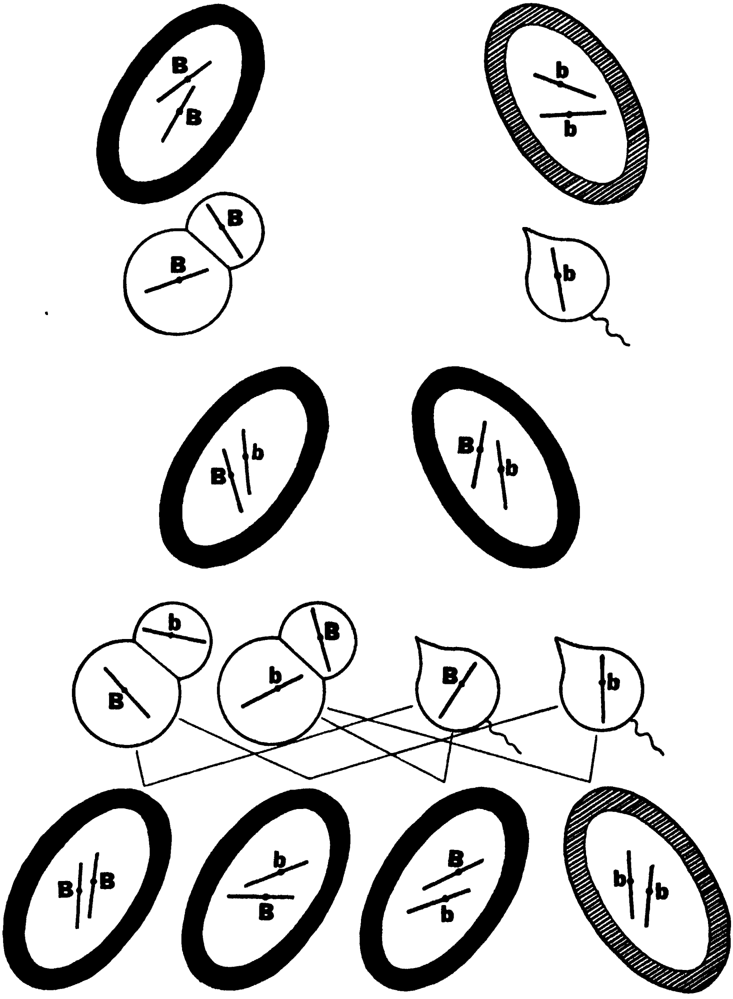


Fig. 42.—Diagram explaining the inheritance of color in mice. Black-bordered ellipses, black mice; shaded figures, brown mice. Eggs and spermatozoa are distinguished by their shapes. Straight lines in the cells or animals are chromosomes. *B*, gene for black; *b*, gene for brown.

with *b*. Since the pair of chromosomes is placed on the reduction spindle at random, one kind of egg should be about as abundant as the other. In the male, each reduction division yields one

cell with B and one with b, hence there are two kinds of spermatozoa, equally numerous.

The two kinds of eggs are fertilized by the two kinds of spermatozoa at random. Consequently the four combinations should be about equally frequent. Fertilized eggs of genotype BB and those containing Bb all produce black mice. Only those possessing the genotype bb yield brown animals. The F_2 generation thus consists of approximately three-fourths black and one-fourth brown mice.

Uncertainties in F_3.—When an F_3 generation is bred from these F_2 animals, the uncertainties due to dominance appear. While the brown mice are known to be bb, there is no way to tell whether a black one is BB or Bb. A mating of two F_2 brown animals yields only brown offspring; but two black individuals from F_2 may yield only black offspring (if at least one of the parents is BB), or they may produce both blacks and browns (if both parents are Bb). In the latter contingency the black and brown offspring are in the ratio of about 3:1, as in the F_2 generation. Other types of matings involving the F_2 mice are possible, but every time a black member of this generation is used there is doubt concerning the genotypes of the prospective offspring, for the black parent may be either BB or Bb.

Choice of Symbols of Genes.—In symbolizing genes by means of letters geneticists have adopted a convention which may now be presented, since dominance has been described. An initial letter is ordinarily used, unless it has been pre-empted for some other pair of genes. Of the two characters which are contrasted, the newer one, in the history of the race, suggests the symbol. In snapdragons the letters *Ii* were chosen in the belief that ivory flowers are a more recent development in the evolution of these plants than are red flowers. For color in cattle the letters *Ww* were selected on the supposition that red cattle have existed longer than white ones. In mice the letters *Bb* cqme from brown, rather than black, since brown is presumably the newer character.

In the above examples the relative ages of the two characters are not directly known. In breeding experiments the new character sometimes arises by mutation, under observation, and then there is no uncertainty in the application of the convention. When the red eye of the vinegar fly Drosophila, much

used in genetic studies, mutated in one individual to a vermilion eye, the symbols of the genes were *Vv*. When the body color of one of these flies mutated to yellow (from gray), the symbols chosen for the genes were *Yy*.

If one of the contrasted characters is dominant over the other, the capital letter is used for the dominant gene and the small one for the recessive. In the color of mice *B* accordingly designates black, *b* stands for brown. Vermilion eye in Drosophila, being recessive, is symbolized by *v*, the wild-type red eye by *V*. When neither character of a contrasted pair is dominant over the other, the allocation of the capital and small letters is optional. There is no particular reason why *I* should represent red color in snapdragons; it might quite as logically denote ivory.

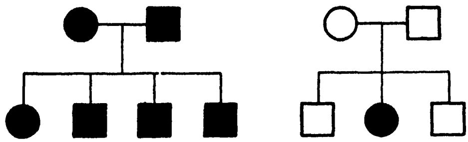


FIG. 43.—Family histories revealing the mode of inheritance of brown and blue eyes in man. Circles are females, squares males. Black symbols, blue-eyed; white symbols, brown-eyed. Blue is shown to be recessive, brown dominant.

Recognizing Dominance in Human Pedigrees.—When heredity is being studied in laboratory animals which are inexpensive to rear, it is usually possible to make the first cross with animals which are known to be homozygous for their respective characters. If the F_1 generation is of the same phenotype as one of the parents, the character it exhibits is dominant, and the character of the unlike parent is recessive.

In human heredity it is not so simple to ascertain which character is dominant. It is impossible to inbreed stocks for several generations to insure that all individuals are homozygous at the outset. Moreover, the "experiments" themselves are not carried out at the will of some breeder; one has to take the information that is available in accessible family histories. In addition, families are small, so that a group of brothers and sisters who should theoretically divide in the ratio of 3:1 between two phenotypes may easily not include the minority class at all. It is necessary, therefore, to judge the heredity of a given character from many family histories, in which the parents are likely to have different genotypes. How are dominance and recessiveness best judged under these circumstances?

The easiest judgment is derived from families in which the parents are alike, and the children are numerous enough to include both classes if two classes are to be expected. Examples will best illustrate the principle. In family histories, females are represented by circles, males by squares. Lines connecting the symbols readily indicate the family relationships. The symbols are usually blackened for individuals who exhibit some unusual character, such as extra fingers, and left white for the "normal" individuals. When the contrasted characters are

FIG. 44.—Symphalangy, or stiff fingers due to fusion of the bones at some of the joints. The fingers are not usually shortened appreciably. (*From Hefner in Journal of Heredity.*)

both common in people in general, either one may be blackened. In any case, a key to the figure must explain the symbols.

By means of such symbols the inheritance of brown and blue eye is represented in Fig. 43, in which the blue-eyed members of the family are represented by black. In both families the two parents are alike. In the family at the left, both parents are blue-eyed, and all the children are blue-eyed. While a single family of this sort would hardly suffice to indicate the mode of inheritance of blue eyes, if many such families are found, they would show that blue eye is recessive. When both parents exhibit the recessive character, all their children must show it.

The best single test for dominance and recessiveness is shown at the right in Fig. 43. Here the parents are both brown-eyed, and one of their children is blue-eyed. In such a family, the

parents must exhibit the dominant character (they are heterozygotes), and the child that differs from them possesses the recessive character.

A very common method of discovering whether a character is dominant is to note whether it occurs in every generation in the direct line of descent, or whether it skips a generation

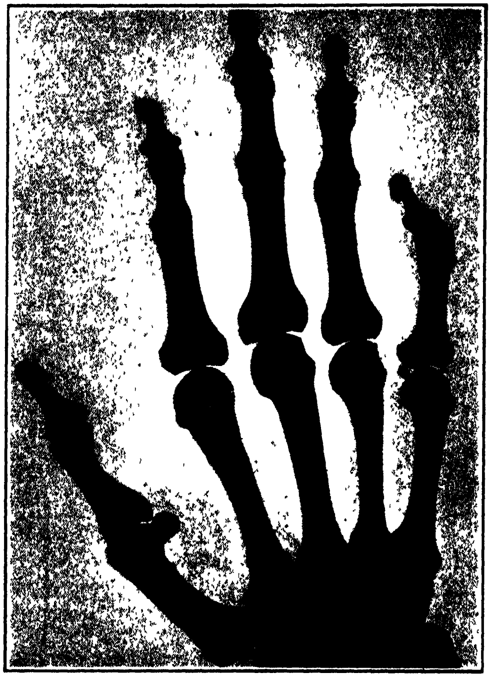


Fig. 45.—X-ray photograph of the right hand shown in Fig. 44. The bones at the first joint beyond the knuckle are fused in each finger. (*From Hefner in Journal of Heredity.*)

now and then. This test is here applied to symphalangy in a known family history. In this character the fingers are stiffened by the fusion of the segments of bone at the joints. The hands in Fig. 44 have the basal two phalanges of each finger thus joined to form a long segment. That there were two bones in such a segment, but that they were united in growth, is indicated by the X-ray photograph of one of these hands in Fig. 45.

A family history in which many members are symphalangic is given in Fig. 46. The record has been abbreviated by including

the marriages of only some of the symphalangic persons. It will be noted that in each family in which there are one or more stiff-fingered children, one of the parents is likewise symphalangic. Since this is true in every instance, the direct line of descent includes one affected person in every generation. This is in general the mark of a dominant character, for wherever one child shows a dominant character at least one of its parents must also show it. It would be possible for a recessive character

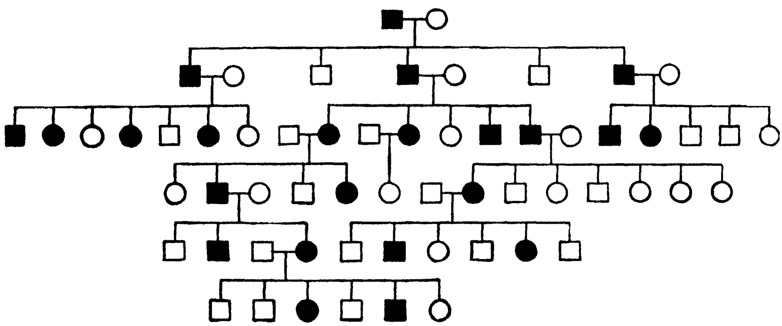


FIG. 46.—Family history of symphalangy. The character is shown to be dominant.

to appear in each of several successive generations in the direct line, provided that the persons marrying into the family are either recessives or heterozygotes; but every-generation appearance could not be universal for a recessive character. Occurring in every one of even a few successive generations would be very unlikely for an uncommon character, for then the persons marrying into the family could seldom be even heterozygous. Contrasted with the occurrence of a dominant character in every generation, a recessive character is regularly absent from some generations in the direct line.

All these criteria of dominance and recessiveness are based on the assumption that inheritance is simple, unmodified by the action of other genes. As will appear in later chapters, many characters are noticeably affected by more than one pair of genes; and even dominance may be modified by other genes.

CHAPTER IX

BACKCROSS AND TESTCROSS

In the preceding chapters the illustrative crosses have been described as passing to an F_2 generation after the F_1. This is a very common type of experiment, and there are reasons for its prevalence. When a new character has just sprung into existence or has first been discovered, there is no stock of individuals of that kind upon which to draw; there is just one individual. In these circumstances the new type can only be crossed with the "normal" or unmutated form; and to mate together their heterozygous offspring is the quickest way to get a stock of individuals showing the new character. If neither of the characters involved in the cross is new, and there are abundant bearers of each one, the F_2 generation is still a very advantageous one, because there is no need to secure virgin females from the F_1 generation in order to produce it. The male and female members of the F_1 generation are together from the first, and any random matings made in advance are precisely those which the breeder would make if he chose to control them. Hence, from F_1 to F_2 is usually the easiest course.

An F_2 generation is not as a rule, however, the most instructive one. If the experimenter's object is to discover how the characters are inherited, and he has stocks of individuals exhibiting them on hand, or if the mode of inheritance is already known and he wishes only to know the genotype of certain doubtful individuals, other crosses are much more useful.

Backcross.—A common procedure in experiments is to make what is called a backcross, that is, a mating of an F_1 individual with one of its parents or with an individual having the same genotype as one of the parents. The backcross may be made to either parent, as shown in Fig. 47. The contrasted characters in this illustration are (1) the smooth coat of guinea pigs, in which the hairs on the back and sides of the body all slope in the same general direction, backwards, and (2) rough coat, in which there are whorls of hair radiating like spokes of wheels at several

places on the body, causing elevated tufts where hairs of opposite slopes meet. As the central animal (F_1) in the figure shows, rough coat is dominant; smooth is recessive.

This heterozygous F_1 may then be mated with either of the parent types. Both matings are backcrosses. If the F_1 animal is mated with a smooth one (left, in the figure), two kinds of offspring, rough and smooth in about equal numbers, are produced. If the F_1 is mated to a rough animal (right), homozygous like the rough parent, all the backcross offspring are rough, although, as is indicated below, they are of two genotypes about equally numerous

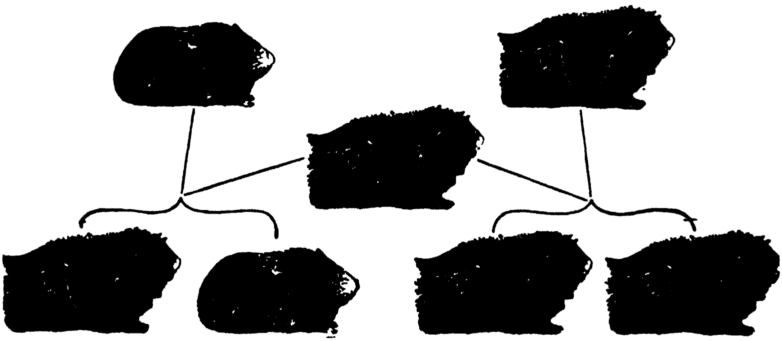


FIG. 47.—Backcrosses, illustrated by inheritance of smooth and rough coats in guinea pigs. Rough coat is dominant. Backcross to recessive parental type (left) yields two phenotypes, but to dominant parental type (right) only one phenotype.

Explanation of Backcross.—The explanation of the results of the two backcrosses is shown in Fig. 48. The general purport of the chart will be understood from the scheme used in earlier figures. In the two matings of the F_1 generation, it is necessary to depart somewhat from the plan of Fig. 47, which the chart illustrates, for, since the F_1 is represented only once (as a female), the animals to which it is mated must both be males; hence the two parents would not be permissible. However, male animals of the same genotypes as the parents are used. On the left, the backcross results in two types of offspring, rough and smooth, in equal numbers. The backcross on the right yields only rough offspring, but they are of two genotypes, *RR* and *Rr*, equally numerous.

The 1:1 ratio, whether of phenotypes or genotypes, is characteristic of the backcross. The two classes are equally abundant

because the two kinds of eggs (or spermatozoa) produced by the F_1 animal are equally numerous. The ratio of offspring is directly dependent on the ratio of germ cells produced by the heterozygous parent. It is important that these two ratios (offspring and gametes) be mentally connected, for later it will

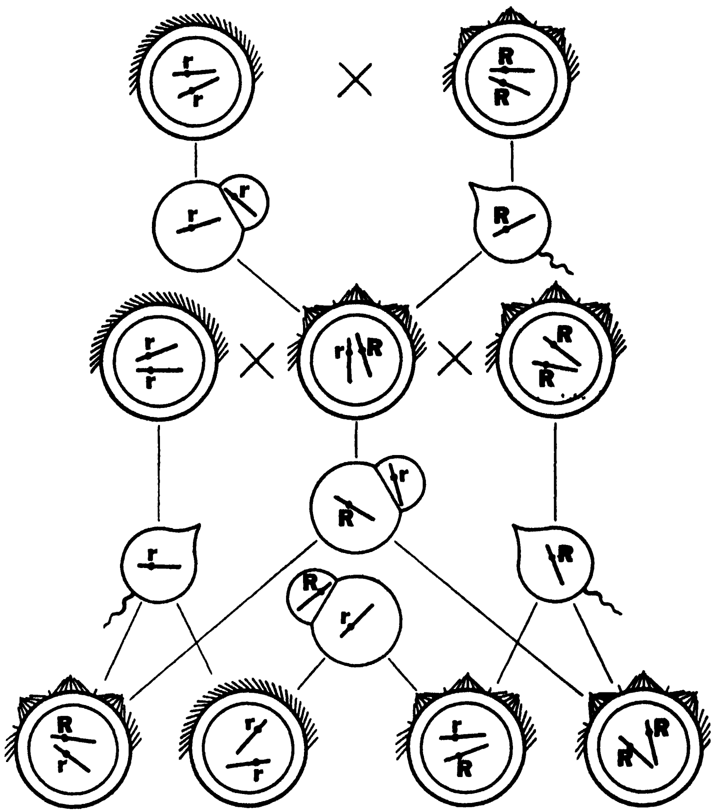


FIG. 48.—Diagram illustrating inheritance of smooth and rough coat as in Fig 47. Uniform slope of lines at periphery of circles indicates smooth, variable slope rough coat. *R*, gene for rough coat; *r*, gene for smooth coat.

be necessary to infer some irregular ratios of gametes from irregular ratios of offspring. They will be the same ratio in every such instance.

Of the two possible backcrosses, only one is usually made. That one is between the F_1 and the recessive parent type. This cross yields two kinds of offspring visibly different, that is, two phenotypes. The other backcross yields only one pheno-

type, and there is no way to tell which individuals are heterozygous, which homozygous. The backcross to the recessive is thus the more informative of the two.

Testcross.—The essential feature of this more usual backcross is that it is a mating between a heterozygote and a recessive homozygote. Now, such a mating is not always a backcross, because one of the parents is not always recessive; the heterozygote involved may, for example, be one of the offspring of two heterozygotes, as Aa derived from $Aa \times Aa$. The parentage of the heterozygote may even be unknown. Yet in neither of these circumstances is the significance of the cross between a heterozygote and a recessive homozygote any less because it

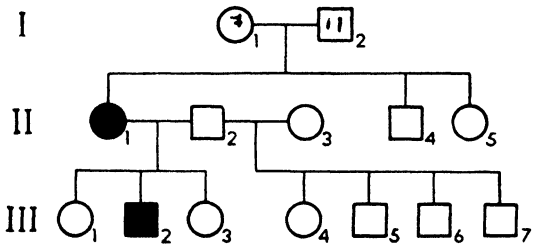


FIG. 49.—Pedigree of feeble-mindedness in an actual family, to illustrate method of determining genotype of a phenotypically dominant individual.

happens not to be a backcross. Moreover, the mating of a dominant individual to a recessive one may be made for the purpose of determining whether the dominant one is a heterozygote or homozygote, in instances where its genotype is not known. Indeed, this latter type of cross is much the most common; phenotypically dominant animals or plants are mated to recessive ones to ascertain their genotypes. If the offspring from such a mating are part dominant, part recessive, the dominant-appearing parent is heterozygous; if the offspring all show the dominant character, the dominant parent is homozygous.

The mating of a phenotypically dominant organism to a corresponding recessive is accordingly known as a *testcross*. The testcross is more valuable when several pairs of genes are simultaneously studied, because the same labor yields more information. It is particularly useful when some of the pairs of genes are in the same pairs of chromosomes. These are topics for later chapters.

Judging Human Genotypes.—The principle of the testcross is used repeatedly in determining the genotypes of certain people,

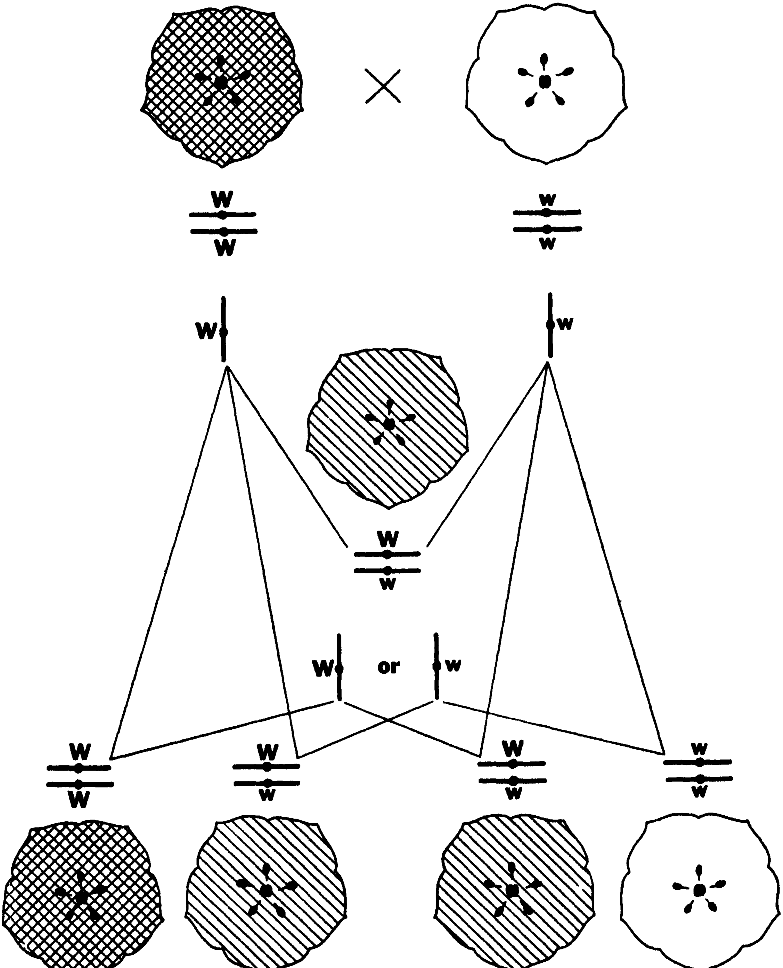


FIG. 50.—Inheritance of yellow and white color in a backcross in four-o'clocks. Crosshatched flowers, yellow; single oblique shading, pale yellow; unshaded, white; *W*, gene for yellow color; *w*, gene for no yellow (white).

even though these people have not been deliberately crossed to a recessive type. To make such a test, the mode of inheritance of the character in question must already be known. If it be supposed that feeble-mindedness is a simple recessive character,

an assumption that is not quite justified but is nearly correct for certain types of the defect, it is often possible to say that a given individual, who is mentally normal, is heterozygous for the deficiency. This can be done for one individual in the family history shown in Fig. 49. To identify the various members of a pedigree, it is customary to designate the successive generations by Roman numerals, I, II, III, and then give the members of each generation Arabic numerals from left to right. The man II-2, who married a feeble-minded woman, is shown to be heterozygous because his son by that marriage, III-2, is feeble-minded. When this man remarried, this time a normal woman (II-3), the children in his second family have one chance in two of being heterozygous. This is a probability which any one proposing to marry into the family ought to know, and the testcross involved in the marriage of II-1 and II-2 reveals it. Incidentally, the pedigree also shows that I-1 and I-2 were both heterozygous, but it is not a testcross which furnishes the evidence.

Backcross without Dominance.—For the sake of completeness, crosses between organisms heterozygous or homozygous for characters which lack dominance should be mentioned, though such matings need never be made to test the genotype of any individual. If a yellow four-o'clock is crossed with a white one, the offspring are pale yellow (Fig. 50), showing that neither color is dominant. If a pale yellow F_1 plant is pollinated by a deep yellow (hence homozygous) one as on the left of the figure, half their offspring should be deep yellow, half pale yellow (heterozygous). The other backcross, of F_1 to white (right of figure), yields two kinds of offspring, half pale yellow and half white. Each backcross thus results in two phenotypes among the offspring.

Such backcrosses are useful exercises in the elementary study of heredity, as demonstrating that the F_1 organisms produce two kinds of germ cells in about equal numbers. They are not properly called testcrosses, however, since it is never necessary to make any test of the genotype of any individual with respect to a pair of characters lacking dominance; the genotype is in such instances obvious from the phenotype.

CHAPTER X

SEX-LINKAGE

The characters whose heredity has been described in previous chapters bear no particular relation to sex. When a cross was effected, it made no difference which parent introduced which character. The two *reciprocal crosses* (crosses involving the same characters but with the sexes interchanged in the two) would have yielded identical results. A backcross could be made with either sex from the F_1 generation, and two equal classes of offspring would be produced. Also each class in an F_2 or a backcross generation would, if numerous enough, include both sexes, in essentially the same proportion as the sexes occur in the general population.

All this equality or indifference of the sexes in these crosses springs from the fact that *each* sex has two chromosomes containing the genes most closely related to the characters in question. These chromosomes separate in the reduction division in essentially the same way in both sexes, producing two equally numerous kinds of eggs or of spermatozoa. Random union of the eggs and spermatozoa occurs, regardless of which of the alternative genes the cells contain. Under these circumstances, the sexes must behave in the same fashion in transmission.

Chromosomes and Sex.—Although most of the chromosomes in animals and plants are equally represented in the two sexes, one pair of them frequently differs. In man, each sex has 48 chromosomes. Of these, 46 (23 pairs) are essentially alike in both sexes. The remaining two in the female are alike; but in the male, one chromosome of this pair is like those of the female, while the other is much smaller (Fig. 51). The two chromosomes of this pair in the female are known as X chromosomes; the one chromosome of the male which is like them is likewise an X chromosome while its smaller mate is called a Y chromosome. The chromosomes of this pair, which are differently related to the sexes, are known as *heterosomes;* the other 46 chromosomes are called *autosomes.*

The mammals in general are like man in this respect, the females having two X chromosomes, the male an X and a Y, and both sexes being alike in their autosomes. Or the male may have an X but lack the Y. Most of the insects are likewise of this constitution, though it is a little more common in them to drop the Y chromosome altogether, so that females are XX, males simply X. Some fishes are also like the mammals in their heterosomes.

In some other animals a similar situation exists, except that the sexes are reversed. These other animals are the moths,

FIG. 51.—The chromosomes in man. At left, those of a dividing spermatogonium; there are 48 of them. At right, side view of spermatocyte at reduction division; X and Y chromosomes, unequal in size, have gone ahead of most of the other chromosomes to the ends of the spindle. (*From Painter in Journal of Experimental Zoology.*)

caddis flies, birds, and some fishes. In them it is the male that has two similar heterosomes, the female two unlike ones. To indicate this reversal of the heterosome relation between the sexes, American geneticists have usually called the heterosomes of these several groups Z and W, respectively. The male has the constitution ZZ, the female ZW. Sometimes the W chromosome is missing, and the female has simply Z.

A number of plants show a similar relation of chromosomes to sex, but the consequences of that relation, which are pointed out later in the chapter, have not been so generally ascertained. Most of what follows will therefore pertain chiefly to animals.

In all these organisms it should be made clear that sex depends on the chromosome outfit. A fly is a female primarily because

it contains two X chromosomes in each cell, or a male because it has only one X (with or without a Y). A moth is a male because it has two Z chromosomes, or a female because it has only one Z (perhaps with a W).

Heterosomes and Germ Cells.—These pairs of heterosomes behave in maturation essentially as do the autosomes. The X chromosomes of a female or the Z chromosomes of a male come together in a pair, and in reduction go to different germ cells, which therefore contain but one such chromosome. The X and Y in a male fly or mammal or the Z and W in a female bird or moth may not pair, since they have few genes in common and likeness of genes seems to be the reason for pairing; but they do go to different germ cells. Hence, the spermatozoa of a mammal are of two kinds, half of them containing an X chromosome, half of them a Y (or no heterosome at all in species in which Y has been lost). The eggs of a bird are of two kinds, about half of them containing a Z chromosome, half of them a W (or perhaps no heterosome of any kind).

Genes in the Heterosomes.—From these relations of the heterosomes it is clear that, if they contain genes, the characters produced by those genes will be inherited in a special fashion. In the mammals and most insects, females will have two genes of each kind found in an X chromosome, and will transmit such genes in every egg; but males will have only one gene of each such kind unless the Y chromosome contains similar genes, and will transmit them in but one class of their spermatozoa.

These peculiarities in transmission have made it possible to discover genes located in the heterosomes. Literally hundreds of genes for various ordinary characters have been proved to exist in X chromosomes. Only a few are known to be in Y chromosomes; there may be more of them, but if there are they are recessive to any corresponding genes in the X chromosome and cannot come to expression, hence cannot be discovered. The same peculiarities exist in Z and W chromosomes; many genes are known for the Z chromosomes, few for W.

Sex-linked Characters.—Characters produced by genes in the X or Z chromosomes are called *sex-linked* characters. A typical example is that of sable, a black body color, in the vinegar fly Drosophila. If a wild-type female whose body color is gray is crossed with a sable male, all the F_1 generation

is gray in both sexes (Fig. 52). When the F_1 males and females are mated together, the F_2 generation consists of gray and sable approximately in the ratio of 3:1. The sable class, however, includes only males. This latter fact, that the recessive members of F_2 are all of one sex, is what indicates that sable is sex-linked and that its gene is in the X chromosome.

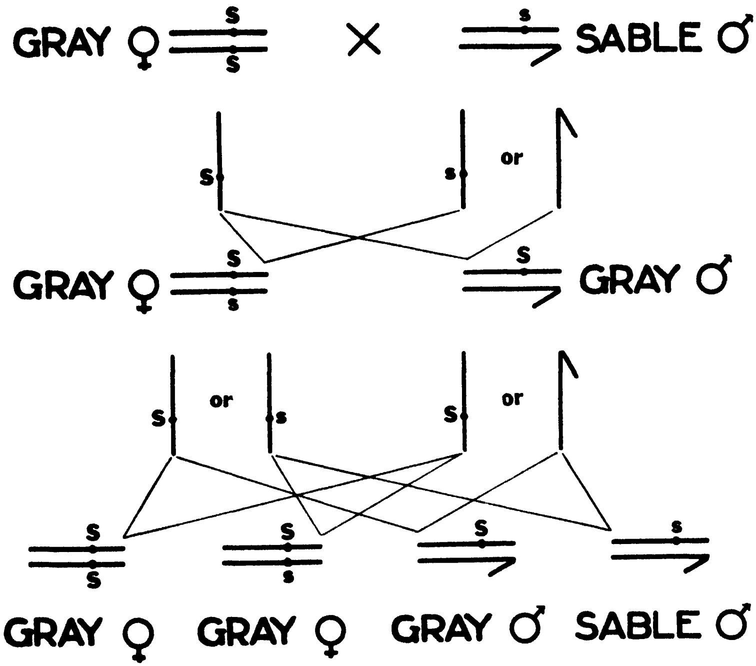


FIG. 52.—Sex-linked inheritance in Drosophila. Mating of gray female and sable male. Chromosomes indicated by heavy lines, genes by dots in them X chromosomes straight, Y chromosomes bent at end. Horizontal chromosomes are those in the diploid cells of flies; vertical chromosomes are in germ cells. *S*, gene for gray; *s*, gene for sable.

How simply this result is explained by assuming that the pertinent genes are in the X chromosomes is shown by the chart in Fig. 52. The heavy straight lines represent the X chromosomes. The Y chromosome is shown bent back at one end, for it is actually J-shaped in this fly. The letter *s* represents the gene for sable, *S* its wild-type alternate (gray). As this mating was made, the female is *SS*, for she has two X chromosomes. The male is *s*, for he has only one X chromosome; the Y chromo-

some has no gene homologous with S, or at least none that is dominant over s. The Y chromosome can therefore be regarded as "empty" of genes in this particular cross.

The X chromosomes are separated by the reduction division, so that each egg receives one, with its gene S. The eggs are represented merely by the X chromosome set vertically. In the male, the X and Y are separated at the reduction division, so that two kinds of spermatozoa are produced. These germ cells are likewise represented by the vertically placed chromosomes. Half the spermatozoa contain the X chromosome, hence the gene s; half of them contain the Y with no pertinent gene.

Fertilization of the eggs by the two kinds of spermatozoa yields two combinations in F_1. One of these is XX as to chromosomes and Ss as to color genes; it yields gray females. The other combination is XY as to chromosomes, merely S in genotype; it produces gray males. The F_1 generation is thus all gray in both sexes.

The F_1 females, being heterozygous, produce two kinds of eggs, and the males, as always in sex-linked characters, produce two kinds of spermatozoa, one with the X chromosome, the other with the Y. The X-bearing spermatozoa in this instance carry the gray gene S. Four combinations in F_2 result from the two kinds of eggs and two kinds of spermatozoa. They are shown in the bottom line of Fig. 52, where their phenotypes are also indicated. The last class in that row consists of sable males—males because they have but one X chromosome, sable because there is nothing in the Y chromosome to dominate over the gene s in the X. Thus a recessive character develops even when only one gene for it is present, because there is no dominant gene to prevent it from appearing.

The Reciprocal Cross.—The preceding experiment started with a dominant (gray) female and a recessive (sable) male. More striking signs of sex-linkage are exhibited by the reciprocal cross, sable female by gray male. The offspring of this cross are not all of one color, but the females are gray and the males sable (Fig. 53), and the F_2 generation, instead of showing a 3:1 ratio, consists of gray and sable in about the ratio 1:1. The two sexes in F_2 are, however, equally represented in the two color classes.

The chart in Fig. 53 explains why these results are obtained. The males in F_1, receiving their single X chromosomes from their mothers, obtain along with them the gene *s*. Hence, since nothing in the Y chromosome is dominant over *s*, these males are sable. In the F_2 generation, one of the XX combinations is *ss* as to genes, and sable females appear, a class not included in the F_2 of Fig. 52.

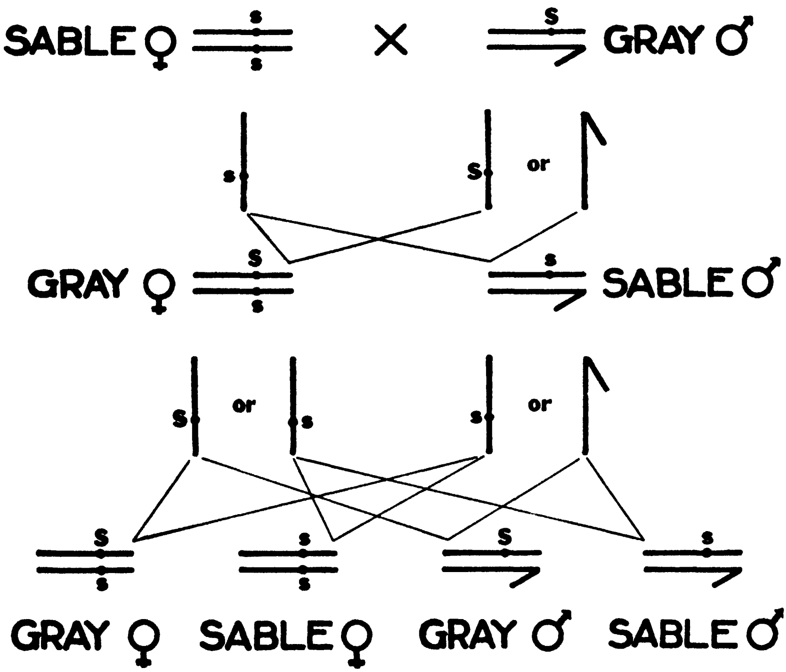


FIG. 53.—Sex-linked cross, reciprocal to that in Fig. 52. The recessive character is introduced with the female, the dominant with the male. Signs of sex-linkage are observed as early as the F_1 generation.

The Marks of Sex-linked Characters.—How is one to recognize sex-linked characters from the results of crosses? In mammals and most insects, in which the females are XX and males XY or XO (no Y chromosome), if crosses between dominant females and recessive males are carried to the F_2 generation and there produce only males in the recessive class, the character is shown to be sex-linked. If the crosses are between recessive females and dominant males, the signs of sex-linkage begin a generation earlier; for the F_1 females show the dominant

character, the F_1 males the recessive character. A further indication of sex-linkage is then found in the F_2 generation from such a cross, for the ratio of dominant to recessive individuals is there 1:1 instead of 3:1. These are the features to be sought in discovering sex-linked characters.

Color Blindness in Man.—Several characters in man are known to be sex-linked. One of them is color blindness, particularly the inability to distinguish reds from greens. Since extensive experiments to determine the mode of inheritance of human characters cannot be performed, it will be valuable practice in the interpretation of family histories to consider how the sex-linked status is assigned to color blindness.

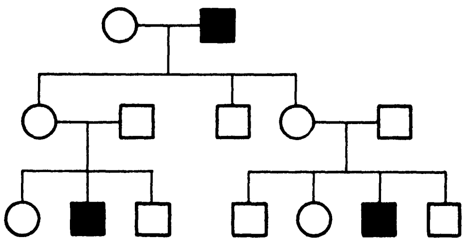


FIG. 54.—Pedigree of color blindness in man, showing that women, though not themselves color-blind, transmit the defect from their fathers to some of their sons.

First, the defect is much more common in men than in women. There are about ten color-blind men for every color-blind woman. This fact by itself is enough to create the presumption that the character is sex-linked, for sex-linked characters in XX-XY (or XX-XO) species normally occur more frequently in males. The reason for this is that a male need receive the gene for such a character from only one parent (his mother) in order to show it, whereas a female, to exhibit a recessive sex-linked character, must receive the gene from both parents.

Second, although women do not often show color blindness, they transmit it as readily as men do. Men transmit the character through their daughters, who do not exhibit it, to half of the sons of those daughters. This skipping of the females and reappearance in some of the males of the next generation is illustrated in the family history of Fig. 54. The two color-blind males of the last generation owe their color blindness to that of their grandfather, though their mothers are phenotypically

normal. This is the behavior of sex-linked characters in species having XY or XO males.

Finally, color blindness rarely occurs in both father and son. A sex-linked character should not appear in both father and son unless the mother also possesses the gene, since males (XY or XO) inherit such characters only from their mothers. In human families a marriage of a color-blind man to a heterozygous woman is rather uncommon, but it does occur. The family history in Fig. 55 includes an example of such a marriage, and, as a result, III-3 is one of the few color-blind men who have a color-blind father. The reason for his color blindness is not his father's color blindness but his mother's heterozygosis. That the mother (II-4) is heterozygous is further shown by her

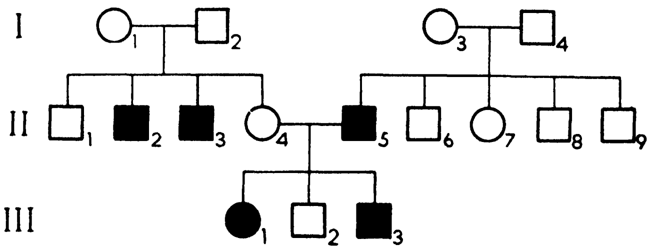


FIG. 55 Pedigree of color blindness, including the rare occurrence of the character in both father and son The reason is a heterozygous mother

color-blind daughter, III-1. It must be inferred, incidentally, that I-1 and I-3 are likewise heterozygous.

Sex-linkage through Z Chromosome.—For an example of sex-linked characters in species whose males are ZZ and females ZW, the ornamental fish Platypoecilus may be used. The inheritance of a black body color, studied by Bellamy (1928), proves that in this species the female has two unlike chromosomes related to sex (that is, she is ZW), while the male is ZZ. The result of a cross between gray and black is shown in Fig. 56. The sexes may be recognized by their lower fins, the posterior lower one of the male being somewhat fingerlike and lying up near the body, while in the female the corresponding fin is a blade projecting down into the water like other fins. In Fig. 56 the female parent is on the left, the male on the right.

The circles beneath the fishes represent the pertinent pair of chromosomes. It is assumed that the W chromosome is smaller than the Z, and the female is shown as WZ, the male ZZ. A black

chromosome indicates that it contains the gene for black color; only the Z chromosomes are distinguished in this way, since the W chromosome contains no detectable gene related to this body color. The male parent is homozygous for black, whereas the female has the gray gene in her Z (larger) chromosome. The male produces but one kind of spermatozoon (not separately shown in the figure); but the female produces two kinds of eggs, one with the Z chromosome and gray gene, the other with the W chromosome with no gene.

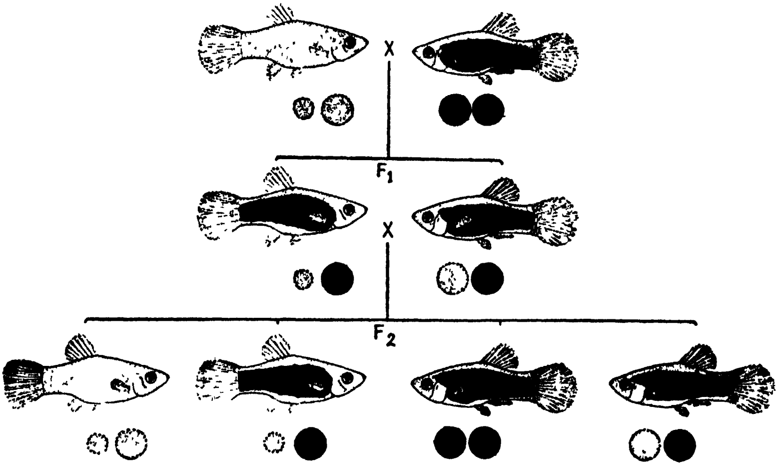


FIG. 56.—Sex-linked inheritance in a ZZ-ZW species, the fish Platypoecilus. Gray female is crossed with black male. Circles below fishes are chromosomes, large ones Z, small ones W. Black chromosomes contain the gene for black, dotted chromosomes the gene for gray. (*Modified from Gordon in The Aquarium.*)

From these germ cells arise two combinations in F_1, which are the two sexes. Since the Z chromosome of the female (left) and each of the Z's of the male (right) contain the gene for black color and since this gene is dominant over gray, all members of F_1 are black, both sexes alike.

The two kinds of eggs and the two kinds of spermatozoa produced by the F_1 animals yield four combinations in F_2, as shown in the lower line of Fig. 56. The two at the right are males because of the two Z chromosomes, the two at the left are females (WZ). Although the ratio of the two colors is $\frac{3}{4}$ black to $\frac{1}{4}$ gray, as in a typical F_2 generation involving any dominant character, it is noteworthy that the recessive fishes

(gray) are all females. This shows that the color is a sex-linked character.

When the reciprocal cross is made (Fig. 57), by introducing the black pattern through the female, the F_1 is divided sharply into two classes, gray females and black males. From them an F_2 is obtained in which the ratio of colors is 1:1, instead of 3:1 as in autosomal characters, and the sexes are equally represented in both classes.

Precisely the same numerical results are obtained as with gray and sable body colors in Drosophila, but the sexes are

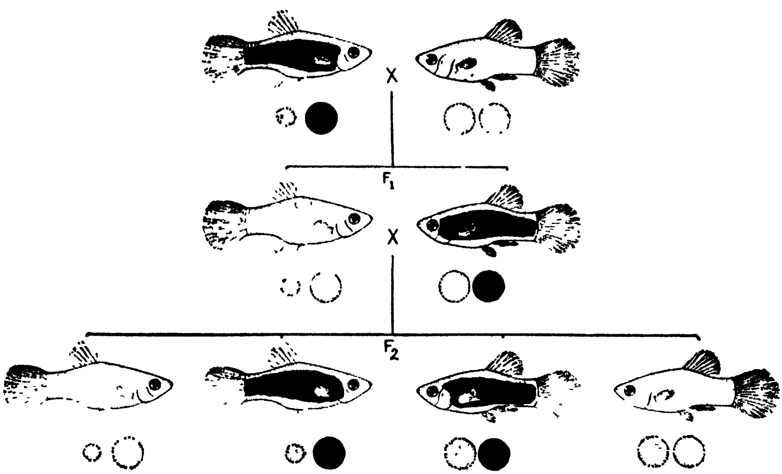


FIG. 57.—Cross in Platypoecilus reciprocal to that in Fig. 56 The F_1 generation shows the body color to be sex-linked, since females are phenotypically recessive, males dominant. (*Modified from Gordon in The Aquarium.*)

reversed in their relation to the mode of transmission. The results obtained in the fly from mating a recessive female and dominant male are obtained in Platypoecilus from mating a dominant female and recessive male, and vice versa. This is the evidence that Platypoecilus has Z and W chromosomes, for the chromosomes are not known from direct observation.

Y-chromosome Characters.—Not always regarded as sex-linked, but inherited in a special way because of the relation of chromosomes to sex, are those characters dependent on genes in the Y chromosome. Relatively few such characters are known. One likely reason for their scarcity is the existence of relatively few genes in the Y. Another reason is that a gene in the Y

chromosome, in order to produce a character, must usually be dominant. There is under normal circumstances only one Y chromosome in each cell, along with an X, so that genes in the Y must be dominant over any homologous genes in the X in order to come to expression.

One character inherited in this fashion is a black spot on the dorsal fin of an ornamental fish, *Lebistes reticulatus* (Schmidt 1920). This example is one of the first to demonstrate Y-chromosome inheritance, and will serve to show that, though some species

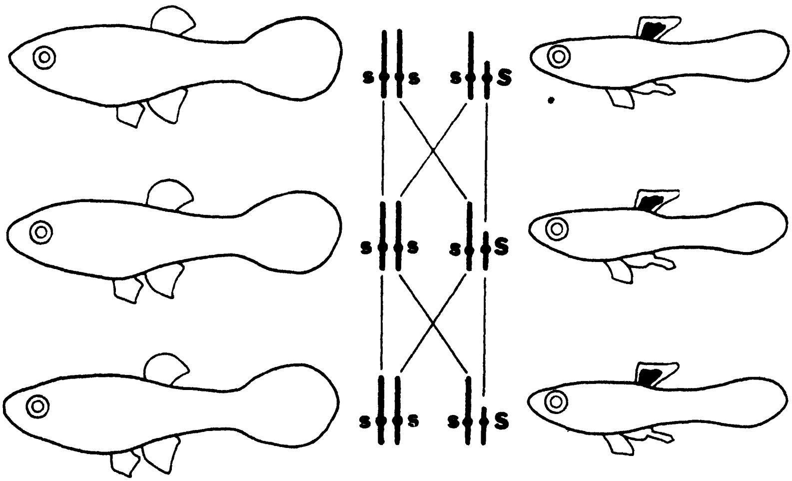


FIG. 58.—Inheritance through the Y chromosome. The black spot on the dorsal fin of the fish Lebistes is confined to males because its gene is in the Y chromosome. X chromosomes are the long heavy vertical lines, Y chromosomes the shorter lines. *S*, gene for dorsal-fin spot; *s*, gene for no spot.

of fish have the ZZ-ZW chromosome outfit, other fish are of the XX-XY type. Lebistes is one having X and Y chromosomes in the male, and the spot in question is transmitted only from father to son (Fig. 58). In this figure the males are all shown on the right, and their Y chromosomes are assumed to be smaller than the X. The separate designation of germ cells is omitted from this figure, but the chromosomes are traced from generation to generation. Every combination of two X chromosomes, which is necessary to produce a female, has only the genes *ss*, and the resulting fish lacks the spot. Every combination of an X and a Y chromosome correspondingly possesses the genes *Ss*, and a spotted male is produced. This mode of transmission

continues as long as the gene *S* is confined to the Y chromosome. Other lines of descent, in which the Y chromosomes have the gene *s* at that locus, produce spotless males. This latter fact, namely, that some males do not have the spot, shows that the spot is not a secondary sex character (see Chap. XXI).

Continuous transmission from father to son in XX-XY species, and only from father to son, is the mark of a Y-chromosome character. In one human pedigree of four generations syndactyly (webbed toes or the side-by-side union of the digits) was found to pass from father to son and not through daughters to their sons. Hence Castle (1922) suggested that the gene may be in the Y chromosome. Other pedigrees of syndactyly should be watched for this relation to the Y chromosome, for some of them do not show it.

Haldane (1937) believes there is evidence that genes for certain human diseases may be located in either the X or the Y chromosome and that they may pass at intervals from the one chromosome to the other (a subject discussed in Chap. XVIII). During their stay in the X chromosome, the characters would behave in ordinary sex-linked fashion; but while in the Y chromosome they would pass only from father to son. The diseases he regards as being thus transmitted are "complete color blindness, xeroderma pigmentosum, Oguchi's disease, recessive epidermolysis bullosa dystrophica, and some cases both of dominant and recessive retinitis pigmentosa." Any gene behaving in the manner described would have to be dominant at least while in the Y chromosome to be detected there.

Transmission of a character from mother to daughter (to all daughters, in fact) would be expected in birds and moths (ZZ-ZW species) if there were a dominant gene in the W chromosome. Such a character may be anticipated, but none seems yet to be on record.

Attached X Chromosomes.—Just as inclusion in the Y chromosome insures transmission of a gene indefinitely from father to son, there is a special situation in which genes in the X chromosomes go exclusively from mother to daughter. This condition is the attachment of the two X chromosomes to each other, so that nondisjunction (page 66) must occur in the reduction division. It arose first in a normal female Drosophila, whose eggs must in consequence contain either the two attached X's, or no X at all.

The attached-X eggs, fertilized by Y-bearing spermatozoa, produced an $\widehat{XX}Y$ combination, which turned out to be female. The no-X eggs, fertilized by X-bearing spermatozoa, yielded X individuals which were males, but sterile. When the $\widehat{XX}Y$ females produced eggs, the attached X's remained together, and the reduced eggs were either $\widehat{XX}$ or Y. Fertilized by the X and Y spermatozoa of a normal male, these eggs entered into the following four combinations:

$\widehat{XX}X$	$\widehat{XX}Y$	XY	YY
♀	♀	♂	
(usually dies)			(dies)

The first combination yielded females, but few of them survived, while the last combination always perished. The surviving offspring were therefore mostly $\widehat{XX}Y$ females and XY males. If these were mated, the same kinds of eggs were produced, and the same combinations formed at fertilization.

The point to be stressed here is that the attached-X chromosomes always went to the females, hence any genes in them were transmitted from mother to daughter.

Gowen (1933) has found an instance of human color blindness in which the character was transmitted from mother to daughter. Since color blindness was already known to be sex-linked, he suggested that the two X chromosomes containing the color-blind genes were joined. Any individual which received them would be both color blind and female, hence the mother-to-daughter transmission.

CHAPTER XI

MULTIPLE ALLELES

In simple genetic experiments one form or condition of a character is contrasted with another form or condition of the same character, as two eye colors, two wing shapes, two color patterns. The genes which are responsible for these two conditions are said to belong to the same pair of homologues. They are so related that in the reduction division of maturation they regularly go to different cells. Each germ cell receives one, but not both.

Alleles.—Two genes which necessarily go to different mature germ cells are called *alleles*. Each is an allele of, or is allelic to, the other. The two conditions of a structure, color, or physiological property which these genes principally help to develop are likewise called alleles. Brown and blue eyes in man are alleles of each other, as are yellow and white flower color in four-o'clocks, red and white color in snapdragons, rough and smooth coat in guinea pigs.

Part of the reason for the separation of allelic genes to different cells is the fact that they occupy the same locus in their respective chromosomes and come together side by side as the homologous chromosomes unite in a pair. Why this identity of locus and pairing of the genes insures that they will go to different cells in the reduction division will be made clear in a later chapter.

Origin of Alleles.—How does it happen that there are different eye colors, different coat colors, and different genes at the corresponding loci of their respective chromosomes to help produce the different phenotypes? They do not always exist. In a young species, one lately arisen from a single ancestry, many of the loci of the homologous chromosomes are occupied each by just one kind of gene. In such a species, chromosome pair 1 in every cell of every individual has, let us say, gene A as the first gene at one end, and gene J as the tenth gene from that end. Chromosome pair 2, we may postulate, has gene T as its twentieth gene, M as its fiftieth, and so on, in every cell in every individual.

Alleles arise by mutation of these genes. As was stated in an early chapter, the genes must be chemical entities of some sort, and mutation is presumably a chemical change. When gene *B* changes chemically, so that in cooperation with the genotype in general it produces a different character, a new gene has sprung into existence at the locus ordinarily occupied by *B*. If the new character is recessive to the old one, the new gene is designated *b*. *B* and *b* are alleles; *M* and *m* are alleles, the one having sprung by mutation from the other. Mutation of a gene produces a new gene in one chromosome, and as a result there are two alleles at that locus.

Multiple Alleles.—Now, genes are probably protein substances, and proteins are of very complex structure. Complex structures have more opportunities to change than simple ones have. Hence, even if a gene is only one molecule of a protein, it probably has the capacity of changing in a variety of ways. A gene should be able to produce by mutation, not just one other kind of gene, but many kinds. It is known, in fact, that this has happened to a number of genes in different organisms. As a result of these unlike mutations, it may occur that the fiftieth gene from one end of chromosome pair 3 in a certain species may be occupied by a number of slightly different genes in different individuals.

Since any individual has two chromosomes of each kind, it may have any two of the genes that have arisen at a certain locus. These two, because they are at the same locus and come together as the chromosomes pair, are forced to go to different germ cells in the reduction division. They are thus alleles of one another. Any one of the genes may be allelic to any other gene at that locus.

Three or more genes occupying the same locus of homologous chromosomes in a species are called *multiple alleles*.

White-eye Series in Drosophila.—One of the largest known groups of multiple alleles is one which concerns eye color in the vinegar fly Drosophila. The wild-type eye color is red. In the year 1910 there was discovered a fly with white eyes, which was later found to be due to mutation of a gene located 1.5 "units" from the "left" end of chromosome 1. In the years of experimentation that have followed, the gene at that locus has mutated again and again. Mostly it has been the wild-type

gene in some individual which has mutated, but occasionally one of the mutant forms of the gene has changed to something else. Some of the eye colors resulting from these mutations have been named eosin, apricot, cherry, coral, buff, tinged, blood, and ivory. In all there have been produced 13 mutant genes at that locus, a recent one having been discovered by Dunn (1935) and called by him "honey." Hence, with the wild-type gene from which they all directly or indirectly arose, there are 14 multiple alleles in this series.

A female fly may have any two of these genes—two eosin genes, or an eosin and a white, or a white and an apricot, or wild-type red and a coral, and so on. In the reduction division the two genes, whichever two they are, separate, one going to the polar body, the other to the oöcyte. Two classes of eggs are produced by a female which has two different genes of this allelic series, just as by any other heterozygous organism. Though only two kinds of eggs may be produced by any one fly, 14 kinds of eggs with respect to this character may be produced by members of the species as a whole.

Symbols of Multiple Alleles.—With more than two genes occupying a given locus in a species, it becomes impossible to use the simple scheme of symbolizing alleles by capital and small letters. A and a would suffice for two of them, but how are the others to be named? A practice has grown up among geneticists of distinguishing the various alleles of a multiple series by superscripts of a common basic symbol. In the white-eye series in Drosophila, since white was the first mutation to be discovered at this locus, the basic symbol is w. The white-eye gene is designated simply by w, but the later mutations are indicated by superscripts. The eosin gene is w^e, apricot w^a, cherry w^c, coral w^{co}, and so on. In harmony with this scheme it is common, though not universal, to call the wild-type gene not W but w^+, the sign $+$ having long been used to indicate the wild-type fly or characters as contrasted with any or all of the mutant characters or genes.

Multiple Alleles in Other Animals and Plants.—Since every gene probably is able to mutate in more than one way, multiple alleles should be common. A number of series are in fact known, several in plants, more in animals. In rabbits color (C or c^+), albinism (c^a), Himalayan albinism (c^h), and chinchilla (c^{ch}) are

members of an allelic series (Fig. 59). Himalayan is like the albino in having pink eyes and white fur, but exposed parts like the ears, feet, tail, and tip of nose are dark brown or black. Chinchillas are silvery gray in appearance because of the absence of yellow pigment in the hair. In addition there are two other alleles of this series.

In mice there are two well-known series (Dunn 1936). One consists of color (C or c^+), chinchilla, which is dilute color (c^{ch}), Himalayan dilution (c^h), and albino (c^a). The other series is

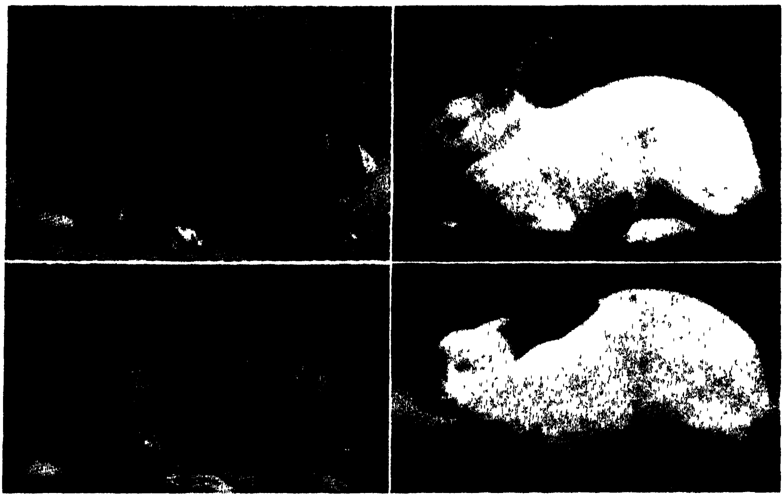


FIG. 59.—Multiple alleles in the rabbit. Upper left, wild type; upper right, albino; lower left, chinchilla; lower right, Himalayan. (*From Castle in Journal of Heredity.*)

made up of yellow (A^Y); agouti (which is gray because the pigments are separated into different regions on each individual hair) with light belly (A^L); agouti with gray belly, as in the wild-type mouse (A); the so-called black and tan, which is black back and light belly (a^t); and nonagouti, or absence of the localization of pigment in the hairs, leaving the coat a solid color, such as black or brown (a).

In ducks Jaap (1934) has found three alleles affecting plumage. These are the mallard, restricted mallard, and dusky types, names which mean more in the young birds than in the adults. The young mallard (M) has a blackish color in the down stage, the dusky (m^d) a lighter general color, and the restricted mallard

(M^R) a black color on head and tail (Fig. 60). The adults have easily recognized differences, which do not, however, readily suggest the names just given. Only the male adults are shown in the figure.

Among plants the snapdragons furnish the largest known series of multiple alleles. There are nine allelic genes representing solid flower colors ranging from red to ivory, besides a red-striped pattern. Corn has some known multiple alleles, as has also barley.

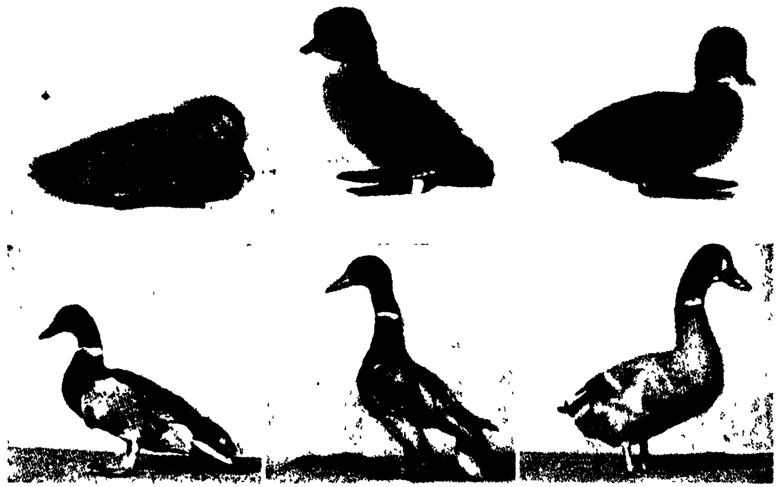


FIG. 60.—Multiple alleles in ducks. Ducklings above, adult males below. Left restricted mallard; center, mallard; right, dusky. (*From Jaap in Genetics.*)

Dominance among Multiple Alleles.—An individual which is heterozygous for the wild-type gene and one of its multiple mutants generally shows the wild-type character. For example, a vinegar fly whose genotype is Ww^e, which means that one of the chromosomes of a certain pair contains the eosin gene and the other the wild-type (red) allele of eosin, is wild type (red-eyed). There are some exceptions to this dominance of wild type among the examples cited in the preceding section; for example, yellow (A^Y) and agouti with light belly (A^L) in mice are both dominant over wild-type agouti (A).

In *compounds*, which are individuals heterozygous for two of the mutant alleles, the dominance relation varies. In Drosophila, the phenotype of a compound is usually intermediate

between the corresponding homozygous mutants. The eosin-white compound (ww^e) has pale eosin eyes, and the apricot-white compound (ww^a) has light apricot eyes. In the albino series in mice (C, c^{ch}, c^h, c^a) the compounds are likewise all intermediate. Dominance is lacking in these instances. However, in the agouti series in mice there is dominance among the mutant alleles, the order of dominance being A^Y, A^L, A, a^t, a, that is, any gene of this series is dominant over any gene following it, with a slight exception as between A and a^t. Thus, A^YA^L and A^YA are both yellow; a^ta is black and tan; and Aa is agouti with gray belly (wild type). But Aa^t, while agouti with gray belly, has a lighter belly than the wild-type mouse has.

When the two genes in a heterozygote help to produce characters that do not interfere with one another, so that both characters could exist in the same individual, it is possible for both genes to be dominant. An example is found in the sections that follow.

Blood Groups in Man.—Certain peculiarities of human blood have received much attention because of their clinical importance. While the red cells of blood float freely and separately in their own serum, it was long ago discovered that red cells from one person introduced into the serum of another person might be agglutinated, that is, collected into little irregular clumps. Not every serum agglutinated the red cells of any particular person, but some combinations led to that result. The reaction was a constant one, for when it was found that the serum of one person agglutinated the red cells of another, every repetition of the test between these same two people gave the same result. Since in blood transfusions it would be serious to introduce blood whose red cells would be agglutinated in the patient, hospitals have had to develop a technique of ascertaining the nature of the blood of both donor and recipient before the transfer is made. That is why so much is known concerning the agglutination phenomenon.

The differences between bloods lie in their possession (or lack) of certain substances called *agglutinogens* in the red cells and certain other substances called *agglutinins* in the serum. There are two of the agglutinogens, designated A and B, with possibly a third which may be ignored until its status is more certain. A given blood may have one, or the other, or both, or neither of them. There are accordingly four kinds of blood, or four *blood groups*. Blood with both agglutinogens in its red cells is said to

belong to blood group AB, blood with A only is of blood group A, that which has only B is of group B, and blood which has neither agglutinogen is said to be of blood group O.

The agglutinins of the serum are likewise of two kinds, α and β. A given serum may have either, or both, or neither of these substances; but their presence or absence has a very definite relation to the presence or absence of the agglutinogens in the red cells. Blood which has agglutinogen A in its red cells *does not* have α in its serum; and conversely, if agglutinin α is in the serum, there is no A in the cells. The two substances are mutually exclusive in any blood. Likewise B and β do not coexist in any blood. These substances are, in fact, the reason for the clumping of the red cells. Bringing red cells with B into serum with β causes the cells to agglutinate; hence no blood could have both. Cells with A would likewise clump in serum with α; consequently no blood could have both A and α. Blood of group AB cannot, therefore, have either agglutinin in the serum, while blood of group O can and does have both α and β in the serum.

From the above it will be seen what bloods can be safely mixed. A and α must not be brought together; so also must B and β be kept apart. Cells of group A would agglutinate in serum of group B or in that of group O, because each of these sera contains α. Cells of group B are agglutinated by serum of either A or O, because each of these sera contains β. Cells of group AB are agglutinated by serum of any of the other groups; but cells of group O are not agglutinated by any other serum.

Clinical Test for Blood Group.—Hospitals use these facts in determining what bloods may be used in transfusion. A simple procedure is the following. Serum of group A and that of group B are kept on hand. A small quantity of each serum is placed on a slide, and a little of the blood to be tested is dropped into each. If neither serum agglutinates the cells, the unknown blood is of group O (Fig. 61, top); if serum of group B clumps the cells but that of A does not, the blood is of group A; if serum A agglutinates the cells but serum B does not, the blood is of group B; and if both sera clump the cells, the blood being tested is of group AB.

Inheritance of Agglutinogens.—Since there is a fixed relation between the agglutinogens and the agglutinins, the inheritance of

blood groups may be described in terms of the agglutinogens alone. It is found that each agglutinogen owes its presence to a dominant gene, so that an agglutinogen is developed if there is

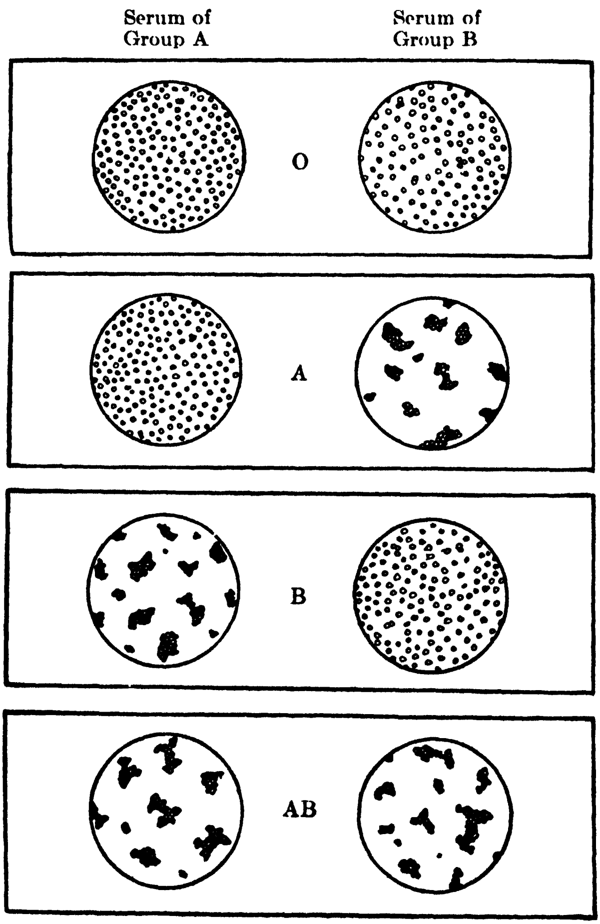


FIG. 61.—Technique of determining group to which an unknown blood belongs. Two drops of serum, one of group A, the other of group B, are put on a glass slide, and a bit of the unknown blood placed in each. If the red cells agglutinate in one, or both, or neither of the drops of serum, the group of the unknown blood is determined in accordance with the scheme illustrated. (*Modified from Snyder, Blood Grouping, Williams and Wilkins Co.*)

even one gene for it. The mode of inheritance was discovered by Bernstein (1925) to be as follows. The genes are alleles of one another, so that one person may have two genes—one gene

twice, or the other twice, or one of each. Now, in addition there is at the same locus in the chromosome in some individuals a third gene which produces no agglutinogen at all. These three genes constitute, therefore, a series of multiple (triple) alleles. Following the usual scheme of symbols, the gene for agglutinogen A may be designated A, that for agglutinogen B is called A^B, while the gene for no agglutinogen is a. Genes A and A^B are both dominant over a, but neither is dominant over the other. Thus, either AA or Aa yields blood group A; either A^BA^B or A^Ba produces group B; aa produces group O; and AA^B produces blood group AB.

Multiple Allelism Probably Common in Man.—As was indicated earlier in the chapter, multiple alleles must be expected to occur frequently, in organisms in general, because of the complexity of genes and their presumptive ability to mutate in a number of ways. Man should be no exception in this regard. Indeed, it is likely that we are confronted continually with examples of multiple alleles, some of which seem to make heredity puzzling. Often a defect appears in different families in somewhat different form, though with some uniformity within each single family. The simplest explanation of such discrepancies is to assume that the same locus of the chromosomes is involved in each case but that the gene is slightly different. Even so apparently simple a character as eye color, which exists in various grades of brown, paling out to blue, may be determined in part by different alleles of a multiple series, though there are other plausible explanations of its variability.

How easily multiple allelism could arise in man will be appreciated from the similarity of two other substances in red blood cells to the two agglutinogens A and B. The new substances are called M and N. They are not clinically important, for there is nothing in the serum to agglutinate the cells containing these substances. They can be detected only by injecting them into the veins of other animals (rabbits, for example) where antibodies are developed in response to the injected substances. By this method it may be discovered whether a given blood contains M or N. The results of such tests show that every person has either M (being homozygous for a gene producing it), or N (being homozygous for a gene producing this substance), or both M and N (being heterozygous for the two genes). M and N are thus alleles

of one another (Landsteiner and Levine 1928). What prevents them from being *multiple* alleles is that, so far as is known, no one lacks both substances. That is, a gene at the same locus which will not produce either M or N has not yet been found. A mere mutation of the gene for M or that for N to a gene that will produce neither substance is all that is required to render the situation of M and N parallel to that of A and B. But then, that is all that is necessary to originate a group of multiple alleles at *any* locus where there are already two unlike genes in different homologous chromosomes. The ease with which this could happen leads to the suspicion that it often has happened and that multiple alleles are common even in man. That so few are known is presumably due only to the difficulty of detecting them where controlled experiments cannot be performed.

It seems likely, therefore, that some of the supposed instances of multiple alleles in man are actually such, though complete proof is not so easy to obtain as in other animals. The uncertainties and lack of agreement are illustrated by studies of color blindness. Waaler (1927) describes four kinds of color blindness, all of them sex-linked as indicated in the preceding chapter. Two of them, he concludes, are represented by genes at the same locus in the X chromosome, and these with the normal gene at that locus would constitute a series of multiple alleles. The other two are likewise due to genes at one locus, but a different locus from that of the first two; these with their homologous normal-vision gene would be another group of multiple alleles. Lenz (1936), however, is not convinced by the argument, and proposes a fractionated gene which is equivalent to two loci with only two (not multiple) alleles at each locus. Similar difficulties are to be expected with other supposed examples in man.

CHAPTER XII

LETHAL GENETIC CHARACTERS

Partly because they modify expected ratios of classes of individuals, partly because of various influences on evolution, lethal genes or lethal situations of other sorts assume a position of some importance. A lethal gene or character or modification of any sort is one which destroys the individual having a certain constitution with respect to it.

Missing Chromosomes.—Several distinct types of genetic situations have the fatal result described. Some of them are

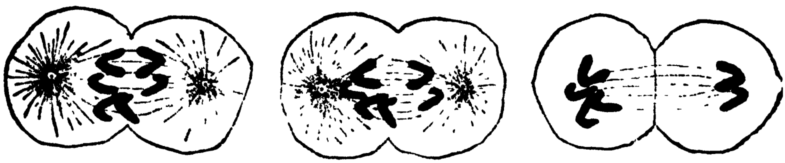


FIG. 62.—Nondisjunction, or the conveyance, to the same cell, of two chromosomes which would ordinarily go to different cells. The lower two chromosomes in each figure are the ones that remain together instead of separating. Division in this case results in one cell having four chromosomes, the other two, instead of each having three.

easily understood. For example, an entire chromosome may be missing. Through nondisjunction (pages 32 and 66), or the failure of duplicated or paired chromosomes to go to different daughter cells at division, a cell may arise which lacks one of the usual chromosomes (Fig. 62). In a diploid cell this would not usually be a serious defect, since the homologue of the missing chromosome is still present, and no kind of chromosome or gene would be wholly lacking. Nondisjunction may happen, however, in the reduction division of an oöcyte or spermatocyte, and, since the mature germ cells are haploid, some of them may as a result lack certain genes altogether. In the germ cells themselves this lack of specific genes usually does little harm in animals, because in them the success of the germ cells is not ordinarily dependent on the contained genes. Even the offspring derived from a germ

cell lacking a chromosome does not necessarily suffer damage, if the other germ cell uniting with the deficient one in fertilization has a complete set. The resulting individual would merely be haploid for one of its chromosomes, diploid for the others. Yet even this condition may change the visible characters of an organism. In Drosophila, for example, nondisjunction in an egg with subsequent fertilization by a normal spermatozoon has produced a fly that lacks one chromosome of the small spherical "fourth" pair (Fig. 63). This fly is accordingly known as haplo-4. It shows its lack of one chromosome by having short

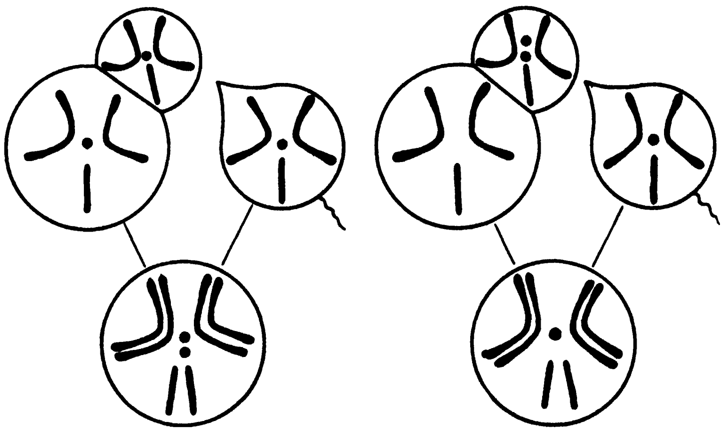


FIG. 63.—Production of haplo-4 in Drosophila, or a fly having only one fourth chromosome. Nondisjunction results in the polar body having two fourth chromosomes, the egg none (right). Fertilization by a normal spermatozoon introduces one chromosome of that pair into the zygote. Normal maturation and fertilization at left for comparison.

blunt wings, paler cross-stripes (Fig. 64), rough eyes, slenderer bristles, slower development, and higher mortality, and it may be sterile.

Because of the higher mortality which it entails, even the haplo-4 condition may be regarded as semilethal. The real lethality of missing chromosomes appears, however, when both chromosomes of a pair are absent. This may easily happen with respect to the fourth chromosome in Drosophila when egg and spermatozoon both lack it. This regularly occurs in a certain proportion of the fertilizations among the descendants of haplo-4 flies. Those fertilized eggs which contain no fourth chromosome at all do not survive.

Deficiencies.—If the total absence of one particular kind of chromosome is fatal merely because some vital part of the organism is thus lost, it should sometimes happen that losses smaller than whole chromosomes would be lethal. Chromosomes are occasionally fragmented, and various things happen to the pieces. They may be turned end about in the same chromosome, they may be attached to an entire chromosome of the same pair, they may be attached to a chromosome of some other pair, or they may be lost in the cytoplasm. In the last two eventualities, they are lost to their original chromosomes. Such losses of fractions

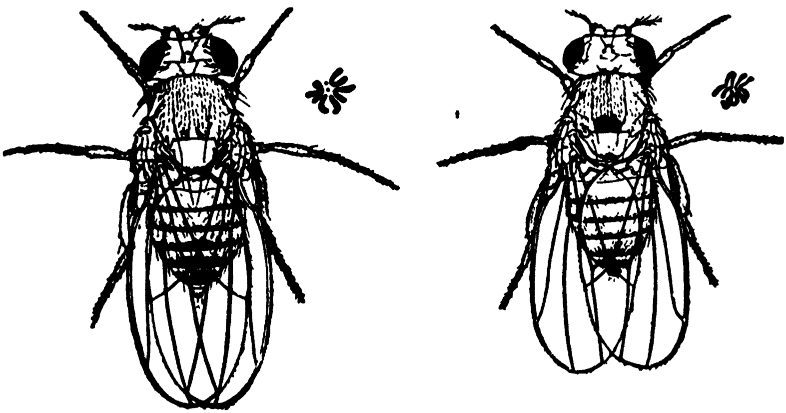


FIG. 64.—Haplo-4 Drosophila (right), compared with the wild type. The group of chromosomes characterizing each is shown beside it. (*From Bridges.*)

of chromosomes, or the gaps where they had been, are known as *deficiencies*.

If a deficiency involves some essential feature of the constitution of an organism, individuals homozygous for it cannot survive. The deficiency may pass down a line of descent through heterozygotes, with homozygotes occasionally being produced and dying. Not all deficiencies are lethal, however. Muller (1935) found a two-gene deficiency in the region of the yellow gene in the X chromosome of Drosophila which was viable even in homozygotes; and Demerec and Hoover (1936) discovered one involving at least four of the bands (salivary gland chromosomes, pages 56–59) at the left end of the X chromosome whose only effect was to reduce the fertility of females homozygous for it, a slightly larger one which was lethal to the whole fly but not to the

cells containing it, and a still larger one lethal to both the fly and a patch of containing cells. In corn, Creighton (1937) found a deficiency which could exist in young plants in homozygous condition, and McClintock (1937) discovered a number of deficiencies that could be homozygous in parts of the plant. If all genes were essential and if each gene occurred only once in each of two chromosomes at the same level, then every deficiency should be lethal. But in the evolution of the chromosomes numerous shifts like those postulated in the preceding paragraph have taken place. Chromosomes have been broken up and their pieces recombined in new chromosomes. Dobzhansky and Tan (1936) have shown such reorganization to have taken place

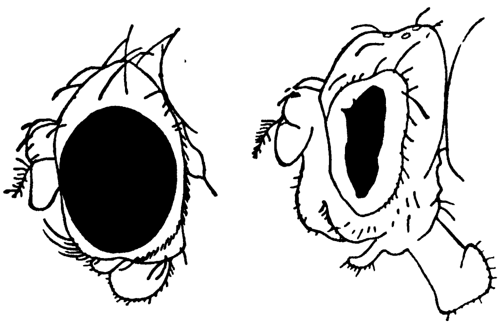


FIG. 65.—Bar eye (right) of the vinegar fly Drosophila as contrasted with the normal eye (left). The area covered by facets is much restricted in the Bar eye and is in the form of a narrow vertical strip. (*From Morgan, Sturtevant, Muller, and Bridges, Mechanism of Mendelian Heredity, Henry Holt & Co.*)

very extensively in one species. Now, in this shuffling and redealing of the genes, it is certain that the same gene sometimes comes to be represented at two or more places, either in the same chromosome or in chromosomes of different pairs. When this has taken place, and then at a later time one of the repeated genes is lost through deficiency, that loss should scarcely prove fatal. Bar eye in Drosophila, in which the area of the eye is limited to a narrow vertical band in place of the large round normal eye (Fig. 65), is according to Bridges (1936) due to a repetition of a group of genes, one group immediately adjoining the other in the same chromosome (Fig. 66), and it has been found that presumed deficiency for Bar is not lethal. That is, one of the repeated groups may be removed, and the fly lives. Its eyes are then like those of the wild type.

Lethal Genes.—A gene need not be absent to kill the organism in which it should exist. It may mutate to some form which is incapable of accomplishing an essential end or which produces a positively injurious effect. Since mutated genes are sometimes dominant over those from which they spring, it is probable that dominant lethal mutations are occasionally produced. They are not usually discovered, however, because the individuals in which they exist perish before the character, if visible, comes to expression. Mutations in general are mostly recessive to their

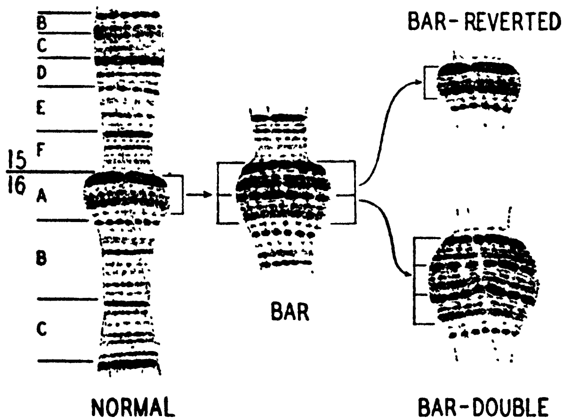


FIG. 66.—The Bar duplication in salivary gland chromosomes of Drosophila. Portion of normal X chromosome at left Most of its segment 16A is repeated in Bar flies (middle). If through irregularities at meiosis one of the repeated segments 16A is lost from Bar, Bar-reverted (upper right) is produced; it is not lethal, since each gene is still represented. Adding a third segment 16A produces Bar-double (lower right), whose eye is still smaller. (*From Bridges in Science.*)

prototypes, and it is presumably true that most lethal mutations are recessive. Recessive lethal genes can be carried along in a stock of organisms, surviving only in heterozygotes, but regularly entering into some homozygous combinations which thereupon die.

Some of the easiest lethal genes to discover are those having two effects—their lethal effect for which they are recessive, and a visible effect for which they are dominant. A classical example is the gene for yellow coat in mice. This gene is a member of one of the series of multiple alleles in mice (Chap. XI) and is symbolized by A^Y. The use of the capital letter in this symbol indicates its dominance with respect to coat color; but in its lethal effect it is recessive. This situation leads to a modified

ratio of the two color classes in certain crosses. Whenever a yellow mouse is crossed with a nonyellow (agouti, for example), two kinds of offspring are produced (Fig. 67). This result shows that one of the mice used was heterozygous, the other a homozygous recessive. If the mode of inheritance of these colors were unknown, it might be assumed that either the yellow or the agouti mouse was the heterozygous one; and, to determine which one, it would be necessary to breed each type further. If two of the agouti mice from the first cross be mated, they yield only agouti offspring, showing that the agoutis were not heterozygous for yellow. But if two of the yellow mice from the first cross are bred, they yield both yellow and agouti offspring. Plainly it was the yellow mouse that was heterozygous. This happens every time a yellow mouse is crossed to one of another color, so that all yellows must be heterozygous.

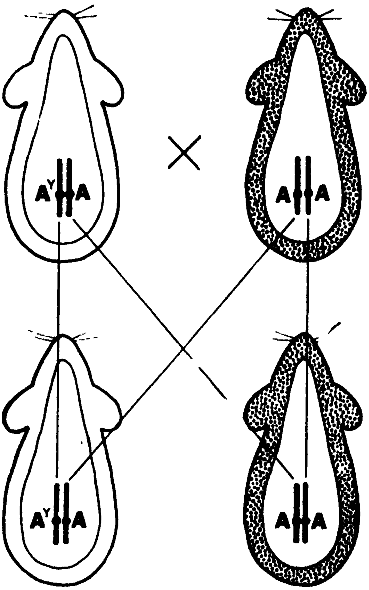


FIG. 67.—Cross between yellow mouse and agouti, showing that one of these mice is heterozygous. Plain figures, yellow mice; dotted figures, agouti. A^Y, gene for yellow; A, gene for agouti.

A yellow mouse may be heterozygous for any of the other alleles of this multiple series. It may contain the genes for yellow and black, or yellow and black-and-tan, or yellow and nonagouti. Every yellow mouse, even in the absence of knowledge of its parentage, may safely be regarded as heterozygous. Why are there no homozygous yellows? It is because the homozygotes die. Suppose two yellows are mated, and that the other gene possessed by each of them is the nonagouti gene a which leads in mice homozygous for it to solid black color. They produce offspring of two phenotypes, yellow and black (Fig. 68), but these are in the ratio of about 2:1 instead of 3:1. Moreover, when the yellows are bred further, they prove to be all heterozygous. There is no class of homozygous yellows. The only

suitable explanation of these results is that the combination A^YA^Y is lethal. The eggs are fertilized, but the embryos die. Abortive fetuses are more common in pregnant yellow mice, and the litters from yellow mothers mated to yellow males are on the whole smaller than those from mothers of other colors, all of which is in harmony with the assumed death of the homozygous yellows.

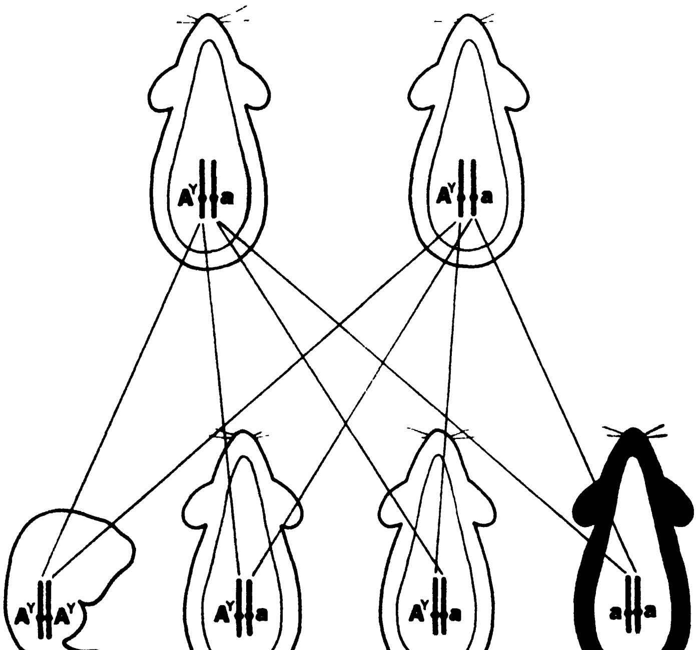


FIG. 68.—Cross between two yellow mice. A^Y, gene for yellow; a, gene for black. Homozygous yellows (A^YA^Y) die in embryonic stage (left, below), leaving ratio of survivors 2 yellow: 1 black.

Other Lethal Characters.—Numerous other animals and plants furnish examples of lethal homozygotes. The "creeper" fowl, a short and crooked-legged type, behaves in inheritance precisely like the yellow mouse. Several different cultivated plants have given rise to chlorophyll-free mutations which are unable to survive. In canaries, a crested type is always heterozygous, the homozygotes dying early. The so-called Dexter cattle show some effect of the Dexter gene in heterozygotes, for these have short legs; but the homozygotes are quite abnormal and are

usually stillborn. A short-tailed mutation in mice has the same effect. The mutant character Dichaete in Drosophila, so-called from missing bristles but more easily recognized by its spread wings (Fig. 69), and Star eye, likewise in Drosophila, in which the facets are disarranged instead of being in regular rows (Fig. 70), both exist only in heterozygotes. In all of these examples the lethal effect is recessive, hence is expressed only in homozygotes; but, except in white plants, some other detectable effect of the gene is dominant, or at least partially dominant, so that the character can be recognized in heterozygotes. In chlorophyll-free plants the visible effect (the absence of chlorophyll) is likewise the lethal effect; it is recessive, and heterozygotes look like normal plants. Here the reason why the lethal

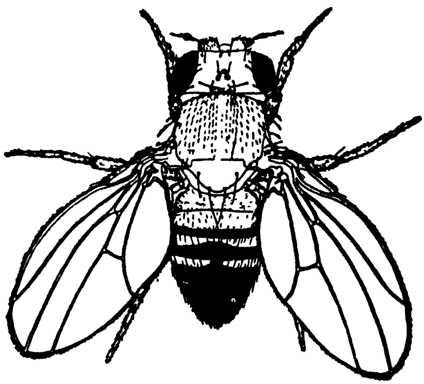


FIG. 69.—Dichæte mutation in *Drosophila melanogaster*. The name refers to the lack of certain long spines on the thorax, but the character is best recognized by the spread wings. Dichæte is lethal when homozygous. (*From Morgan, Bridges, and Sturtevant in Bibliographia Genetica.*)

character has been discovered is that young plants can live for a time on the food stored in the seed; when that is exhausted, they are unable to manufacture their own.

Possible Lethals in Man.—It has been suggested by Bauer (1922) and by Davenport (1930) that hemophilia in human beings may be lethal in the homozygous condition. Hemophilia is a tendency to bleed freely from slight wounds. Extraction of a tooth is for a hemophiliac a dangerous operation. The character is due to some defect, presumably a chemical one, of the blood which prevents or diminishes clotting. It has long been known to be a sex-linked character and is ordinarily recessive, hence is much more common in men, who require only one gene, than in

women, who must inherit it from both parents in order to possess the defect. It may be not quite, or not always, recessive, for heterozygous women have been shown sometimes to have a slower coagulation rate than do homozygous normal women, and in one instance a heterozygous woman was herself a bleeder. Some caution is needed, however, in judging the meaning of such facts, for there are other causes of bleeding which clinically resemble hemophilia.

The significance of hemophilia in connection with lethal homozygotes arises out of the scarcity of hemophilic women. A sex-linked character should, if common enough, appear in both sexes. For a given frequency of the gene in a population, there should be a certain proportion of males and a certain smaller proportion of females exhibiting the character. If the number of

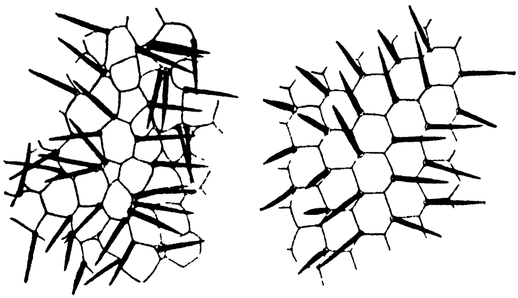


FIG. 70.—Star eye mutation in *Drosophila melanogaster*. The facets and spines disarranged (left) as compared with the normal eye (right). (*From Morgan, Bridges, and Sturtevant in Bibliographia Genetica.*)

hemophilic men be used to determine statistically how prevalent the gene must be, it is then found that the women fall far short of the expected number. Baur and Davenport sought to explain the smallness of the number of hemophilic women by supposing the gene to be lethal in the homozygous state. The scheme as portrayed by Davenport is shown in Fig. 71. The mere bleeding itself might prove fatal to adult women, but the scarcity of hemophilic women extends to the childhood years. The lethal effect must therefore be assumed to operate in some other way than bleeding to death.

One possible obstacle to the assumption of a lethal effect is the fact that the hemophilia gene is not lethal in males. Ordinary recessive sex-linked characters show as readily in males

(with their one gene) as in homozygous females. Furthermore, the experimentally best-known recessive sex-linked *lethal* characters *are* lethal in the male, as well as in homozygous females. Hence, unless there is more to the production of hemophilia than

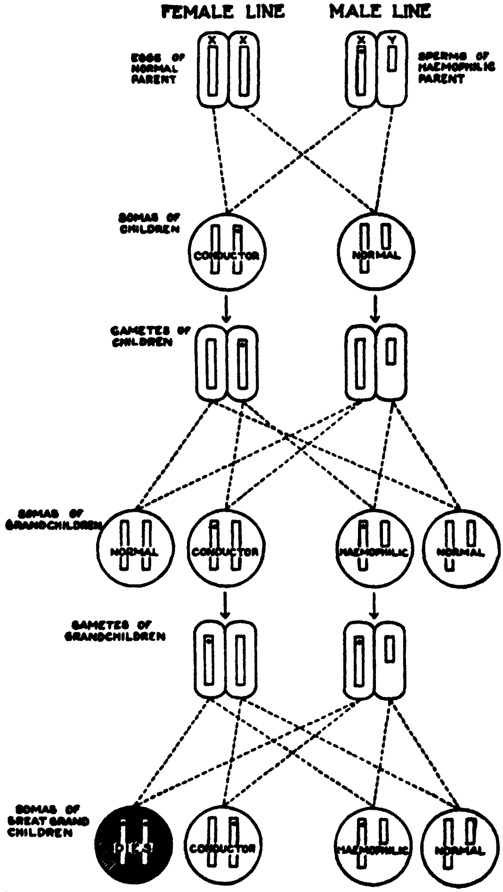


FIG. 71.—Scheme of inheritance of hemophilia in which the character not only is sex-linked, but is assumed to be lethal when homozygous in females. The gene for hemophilia is represented by a dot in the upper ends of certain X chromosomes. (*From Davenport in Genetics.*)

just the gene for it, hemophilic males should perish too. It might be assumed as an explanation of the viability of the males that something in the male physiology, perhaps a sex hormone, counteracts the supposedly lethal effect in that sex, though not counteracting its inhibition of clotting. Or it could be assumed

that women homozygous for the hemophilic gene do exist in the expected numbers and that something in the female physiology, perhaps a sex hormone, counteracts the effect on coagulation and so prevents bleeding. All sons of such women, not merely half of them, should be hemophilic. Or it may be supposed that, unlike other sex-linked lethals, one gene in the male does not produce so great an effect as two genes in the female. This last is essentially what Bauer and Davenport have suggested.

Lenz (1936), however, rejects the statistical evidence that homozygous women are unduly scarce and insists on specific evidence from family histories. He points out that the lethality of the homozygous state would only be proved if a hemophilic man marries a heterozygous woman, and they produced (without interference by an inhibiting hormone or other agent) some nonbleeding but no bleeding daughters. Such family histories he holds have not been recorded.

A particular type of brachyphalangy, shortened second segments of the second fingers and toes, may also be lethal in the homozygous condition. This character is ordinarily regarded as a dominant one because it appears in every individual which transmits it—hence in every one which has the gene for it. Sometimes it is considerably weakened in expression, for reasons to be explained in a later chapter, but it has always been found in a person known to be heterozygous. Now, to get a person who is homozygous, it is necessary that both parents be brachyphalangic. Such matings must be rare, and for a long time none was known. However, Mohr and Wriedt (1919) describe a cousin marriage of this sort, part of a much larger family history which includes 33 brachyphalangic persons. From this cousin marriage came three children (Fig. 72), one of them short-fingered and one a cripple without fingers or toes and with a very much disordered skeleton in general. The latter child lived only a year. Mohr and Wriedt suggest that it was homozygous for brachyphalangy, a condition to be expected in one-fourth of the children of such parents

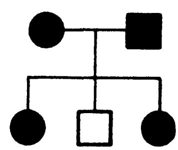


FIG. 72.—Family history of brachyphalangy, part of a much larger pedigree. The parents were cousins Their last daughter, supposedly a homozygote, died in early childhood. (*After Mohr and Wriedt.*)

If this child was a homozygote, its nature illustrates one interesting point besides the lethality of homozygosis. That is

that brachyphalangy is not dominant in the usual sense. To say that a gene or character is dominant should mean that a heterozygote is identical in appearance with an individual homozygous for the gene. In the absence of any homozygotes of the one kind, we are not able to make the necessary comparison. When under these circumstances we can recognize a heterozygote as distinguished from the one known type of homozygote, we call the character dominant. If, however, the quality is expressed in some other, perhaps greatly exaggerated, form in a homozygote, it is scarcely correct to say that the gene is dominant. It is no more dominant than is red or ivory color in snapdragons, or than red or white in Shorthorn cattle. The same may be said of Dichaete and Star eye in Drosophila, for no one knows what flies homozygous for these genes would look like. Yellow color in mice might be very different in a homozygote; no one knows. One could say of these characters that they are at least partially dominant, which is one way of expressing lack of dominance as in pink snapdragons and roan cattle. It is only in those instances in which the homozygote lives to some stage in which its character may be observed that it is possible to say whether the lethal gene is really dominant or not. If Mohr and Wriedt's interpretation of the skeletally abnormal child in Fig. 72 is correct, brachyphalangy is not dominant, nor is it recessive; dominance is lacking as between this condition and normal fingers.

A summary of the action of lethal genes in man and other animals is given by Eaton (1937).

Time and Manner of Action of Lethal Genes.—The contrasts just made reveal some importance of the time at which lethal genes act. Early action, before the pertinent character develops, leaves us in ignorance of its dominance or lack of dominance. Late enough action permits dominance to be ascertained if it exists.

Lethal genes vary greatly in this respect. In plants a number of lethal genes are known to destroy the germ cells in which they occur, and these are known as gamete lethals. Among the zygote lethals (those which act later than fertilization of the egg), the time of destruction may be early or late. Dichaete and Star eye in Drosophila kill the early larvae. Creeper fowls when homozygous die in the shell (Landauer and Dunn 1930) at about the fourth day of incubation out of the 21 days which normal

incubation requires. Short-tailed mice die (Chesley 1932) at the tenth day after fertilization (Fig. 73) out of the usual 21 before birth. Homozygous "bulldog" calves are usually stillborn (Wriedt 1930). White seedling plants survive as long as the food stored in the seed lasts, then starve to death. A number of defects in man which regularly shorten life may be regarded as late-acting lethals.

Fig. 73.—Normal embryo (left) of mouse at end of tenth day after fertilization, and that of lethal homozygous short-tailed mouse of same age. (*From Ephrussi in Journal of Experimental Zoology.*)

How the lethal genes work is not yet understood. Ephrussi (1935) has maintained for a long time, by tissue-culture methods, some of the cells of short-tailed mouse embryos, taken shortly before the embryo died. Embryonic hearts continued to beat for two months, and some of the other tissue cells multiplied and differentiated. These results show that not all cells respond to the lethal genes which they presumably all contain. Whether some particular type of cell is thus susceptible or whether some relation between cells of different types is the cause of death remains an open question.

CHAPTER XIII

TWO OR MORE INDEPENDENT PAIRS OF GENES

Since every organism of the higher groups must possess hundreds or even thousands of genes, and since many of these genes may have mutated within the history of the species, it is inevitable that two individuals which mate should frequently differ in more than one respect. Indeed, unless inbreeding or self-fertilization has been steadily practiced, or there is some other special reason why a population is genetically homogeneous, two individuals which mate are more likely to differ in dozens, scores, or even hundreds of ways. If these various differences do not relate to the same parts of the organism, or sometimes even if they do, it is possible to study the inheritance of a number of characters simultaneously. This is more easily done with only two characters than with three or half a dozen, and the results are simpler if the distinct characters are independent of one another in their distribution to the offspring and in their development or expression in the individual.

Independent Characters.—Two characters are independent of each other in their distribution if inheritance of one of them by a given individual does not create a presumption that the other will likewise, or that it will not, be inherited by the same individual. In terms of genes, this independence means that the entrance of a particular one of the alternative genes of one pair into a given germ cell does not favor or oppose the entrance of a particular gene of another pair into the same germ cell. Such independence exists only when the two (or more) pairs of genes are in different pairs of chromosomes. In general—there are some exceptions—the pairs of chromosomes are independent of one another. That is, in the reduction division each pair of chromosomes may take either of two positions on the spindle, and taking one of these positions does not limit the freedom of another pair to take either of its two possible positions purely at random. Consequently, any genes in one pair of chromosomes are inde-

pendent of any genes in another pair of chromosomes. It is only with such independent genes that this chapter deals; the restrictions placed upon different pairs of genes which happen to be contained in the same pair of chromosomes is a subject for later discussion.

Independence of development or expression of different characters means that production of the one does not hinder, or promote, or modify production of the other. Characters are more likely to be independent in expression if they affect distinct parts of the organism, as color of eye and shape of hair, or shape of wings and color of body. Yet even such apparently unrelated characters are not always independent in expression. Interaction between genes of different pairs is likewise a topic for later treatment.

Color and Shape of Summer Squash.—Illustrative of two independent characters are the color and shape of the fruit of summer squashes. Among the various shapes of the fruit are the spherical, in which the height is about equal to the diameter, and a flattened form which is called the disk. Of the colors, the two here chosen are white and yellow. These characters are independent in expression, since either disk or spherical fruits may be either white or yellow. That they are likewise independent in distribution to the offspring is shown by the numerical results of crosses.

Since white is presumably the newer color and disk the more recently acquired shape, the symbols *Ww* and *Dd* are chosen to represent the genes. Being independent in distribution, the two pairs of genes must be in different pairs of chromosomes. In the diagram in Fig. 74 the chromosome pairs are distinguished by showing them as of different sizes, the long pair containing the color genes and the short pair the genes for shape.

The original cross is between a white disk plant and a yellow sphere. The gene and chromosome composition of these plants is shown within the illustrations of the fruit. Their germ cells receive, from the reduction division, one chromosome of each pair, hence are of the composition *WD* and *wd*, respectively, as shown by the vertical lines beneath the fruit. The F_1 fruit resulting from union of these cells must therefore have the genotype *WwDd*, and, since *W* and *D* are dominant, they are white and disk-shaped.

When these doubly heterozygous F_1 plants produce their germ cells and offspring, the first indication is given that the color and shape genes are independent in distribution. As the chromosomes of the two pairs separate in the reduction division, they may be turned either of the two possible ways on the spindle, so that the long chromosome with *W* may, and in some cells does, go to the same end of the spindle with the short chromosome containing *D*, or it may, as in other cells, go to the same end of the spindle as does *d*. The yellow gene *w* may likewise be turned in such a way as to go sometimes with *D* and sometimes with *d*. Four kinds of germ cells are thus produced, *WD*, *Wd*, *wD*, and *wd*, as indicated by the vertical lines (chromosomes) below the F_1 fruit. Because the placement of the pairs of chromosomes on the reduction spindle is purely fortuitous, these four kinds of cells are about equally numerous. The same numerical relations hold for the eggs and the pollen nuclei—four equally numerous kinds of eggs, four equally numerous kinds of pollen nuclei.

In the production of F_2 plants, therefore, 4 kinds of eggs are fertilized by 4 kinds of pollen, making 16 combinations. Some of these combinations are of course duplicates. A simple way of ascertaining the various combinations is to enter them in a checkerboard, or Punnett square, as in Fig. 74. If the eggs are ranged along the top and the pollen nuclei down the left, their combinations may be placed in the squares where the respective columns and rows cross.

By inspection of the combinations, the type of plant that will develop from each of them may easily be ascertained. Any fruit with at least one *W* will be white, while *ww* will yield yellow; any fruit with at least one *D* will be disk-shaped, *dd* spherical. By assembling all with the same phenotype, we find that 9 of the 16 combinations are white disk, 3 of the 16 are white spherical, 3 are yellow disk, and 1 is yellow spherical. The $\frac{9}{16}$ class has both dominant characters, each of the $\frac{3}{16}$ classes has one dominant and one recessive, the $\frac{1}{16}$ class both recessives.

The ratio 9:3:3:1 is characteristic of an F_2 generation when two independent pairs of genes are involved, in each of which one gene is dominant over the other. The ratio may be obtained algebraically by simply squaring the binomial $3 + 1$, that is, by expanding the expression $(3 + 1)^2$. This means that each pair of characters would by itself yield an F_2 generation consisting of

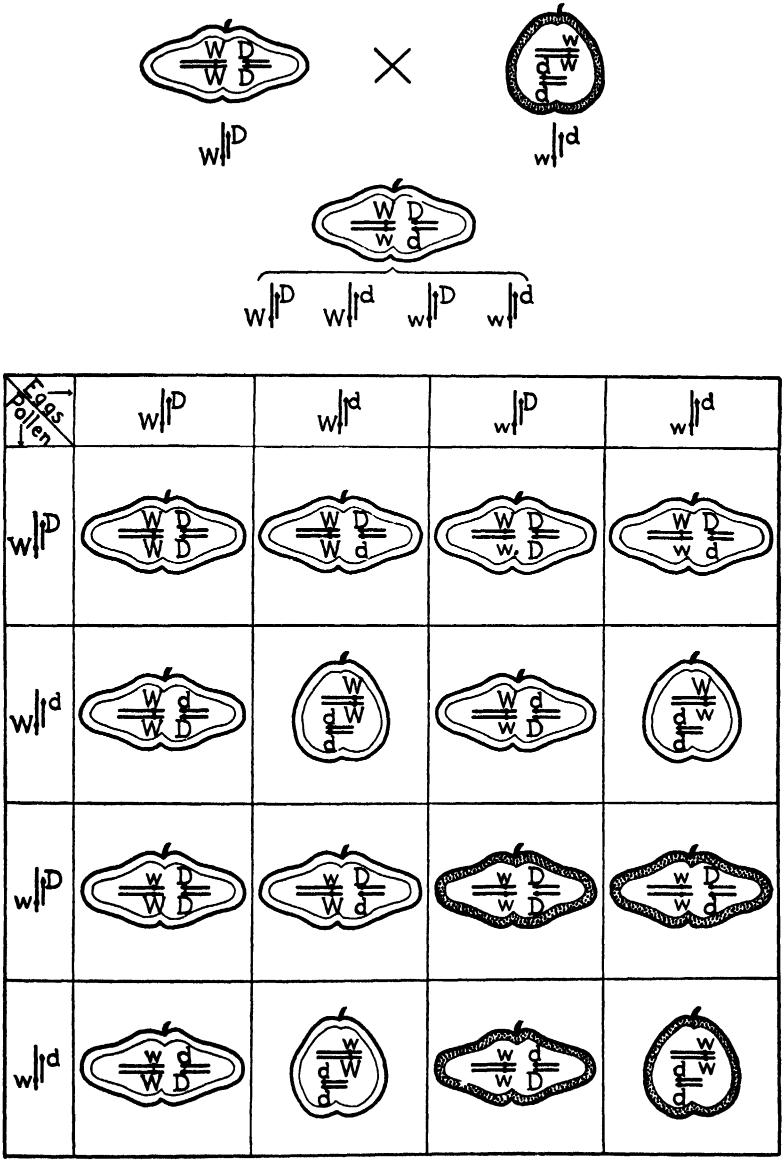


FIG. 74.—Inheritance of color and shape (disk and spherical) of squash fruit. Plain borders, white fruit; dotted borders, yellow. Heavy straight lines are chromosomes, dots in them are genes. *D*, gene for disk shape; *d*, gene for spherical shape; *W*, gene for white color; *w*, gene for yellow.

two kinds of individuals in the ratio of 3:1, and, when the two pairs are combined at random, their combinations are in the ratio indicated by the product of the two separate binomials $(3 + 1)(3 + 1)$. It is these numerical results which indicate that the two pairs of genes really are independent of one another in distribution. Were they in some way restricted in relation to each other, other numbers of the several kinds of plants would be obtained.

The Genotypes of F_2.—Because of the dominance of one gene over the other in each pair, each phenotype in F_2 includes more than one genotype, except the $\frac{1}{16}$ or doubly recessive class (yellow sphere). The 16 combinations thus fall into 9 different genotypes, as follows:

$$
\begin{array}{ll}
\text{White disk} & \left\{\begin{array}{l} 1\ WWDD \\ 2\ WwDD \\ 2\ WWDd \\ 4\ WwDd \end{array}\right. \\
\text{White sphere} & \left\{\begin{array}{l} 1\ WWdd \\ 2\ Wwdd \end{array}\right. \\
\text{Yellow disk} & \left\{\begin{array}{l} 1\ wwDD \\ 2\ wwDd \end{array}\right. \\
\text{Yellow sphere} & \{1\ wwdd
\end{array}
$$

The student may wish to work out an algebraic scheme whereby such a list of genotypes may be drawn up in any specific example without preparing a checkerboard, but it is not important to be able to do so in elementary work.

Testcross with Two Pairs of Characters.—In the preceding cross the independence of the two pairs of genes in their distribution to the germ cells is first demonstrated by the 9:3:3:1 ratio in the F_2 generation. That ratio is a consequence of the equality of numbers of the four kinds of germ cells from each doubly heterozygous (F_1) parent, and this equality of numbers springs from the randomness of combination of the genes.

That the four kinds of germ cells produced by an individual heterozygous for two pairs of genes *are* equally numerous is more directly shown by a testcross. Two pairs of characters in Drosophila may be used to illustrate. One concerns the color of the eye, the normal red eye of the wild-type fly and the mutant brown eye, of which the wild type is dominant. The other

relates to the color of the ocelli, the three small simple eyes on the top of the head. The wild type has reddish-yellow ocelli, the mutant one white, yellow ocelli being dominant. The genes for these two pairs of characters are in different pairs of chromosomes,

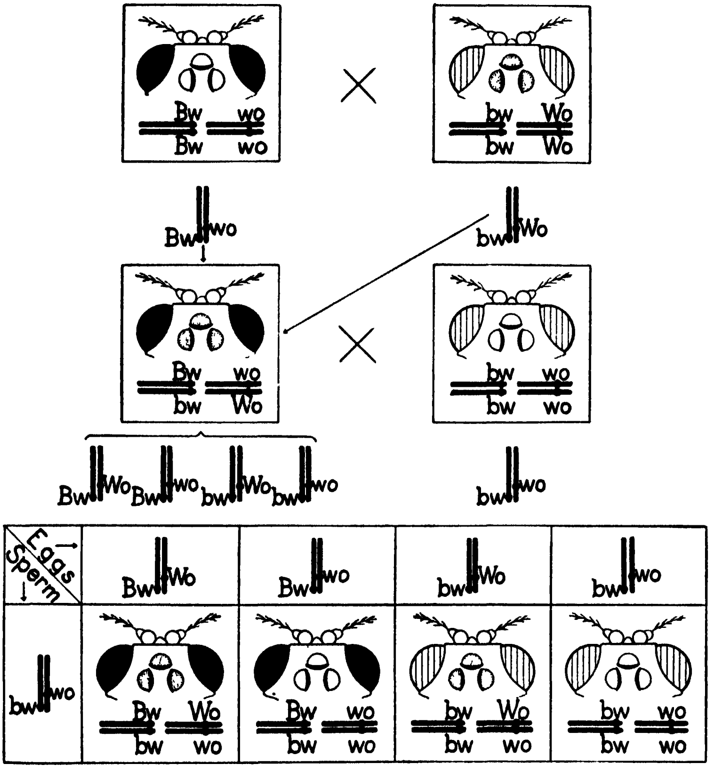


FIG. 75.—Inheritance of color of eyes and color of ocelli in Drosophila. Second mating is a testcross. Genes are represented as in chromosomes. Black eyes are wild-type red, shaded eyes brown; dotted ocelli are wild-type reddish yellow, plain ocelli white. Ocelli are greatly enlarged. *Bw* and *bw*, genes for red and brown eye respectively; *Wo* and *wo*, genes for reddish yellow and white ocelli.

those for eye color being in chromosome 2, those for the ocelli in chromosome 3.

The general scheme in Fig. 75 is the same as in the preceding illustration and requires no extended explanation. Each parent, as there represented, has one dominant and one recessive character, while the F_1 fly has both dominants. The latter is mated with a doubly recessive fly (brown-eyed with white ocelli).

The F_1 fly produces four kinds of germ cells; the double recessive produces only one kind. In the squares beneath are shown the four combinations into which these germ cells may enter.

The four kinds of flies in the testcross generation are about equally numerous. Their equality is a demonstration that the four kinds of germ cells produced by the F_1 fly were equally numerous. And the numerical equality of these germ cells follows from the fact that the two pairs of genes are in different pairs of chromosomes. One is obliged to infer that they are in different pairs in order to explain the equal numbers of the germ cells.

Recombinations in Man.—Similar recombinations of characters in man are frequently witnessed. Since families are small, however, the ratios of the different classes of offspring are less reliable, and it is seldom that one can be sure from a single family history that the genes really are independent of one another. When they are independent, it is inferred that they are in different chromosome pairs. Man has 24 pairs of chromosomes; hence 2 pairs of genes chosen at random have a good chance of being independent.

Recombination, probably independent, is shown by the color and shape of the hair. Hair may be dark or light, the former being approximately dominant. It may be curly or straight, with curliness essentially dominant. Neither of these statements is a whole truth, for both color and shape of hair are inherited in accordance with a more complex scheme, to be described in a later chapter. However, in single families where certain parts of the scheme are eliminated from consideration because all individuals are alike with respect to them, the simple inheritance just indicated may prevail.

An actual family history illustrating the recombination of hair color and hair shape is shown in Fig. 76. The mother had dark curly hair, the father light and straight. Their four children, three girls and a boy, fortunately for this illustration, were of the four possible kinds, light curly, dark curly, dark straight, and light straight. The mother was evidently heterozygous for both characters, while the father was observably homozygous for both recessives. This marriage would serve as a testcross comparable with that in Fig. 75, made for the purpose of ascertaining the genotype of the doubly dominant woman. It was largely acci-

dent that each of the four expected classes was represented in so small a group, but on the basis of the simple and independent inheritance postulated above this should happen in 3 out of every 32 families of the same size from similar parents.

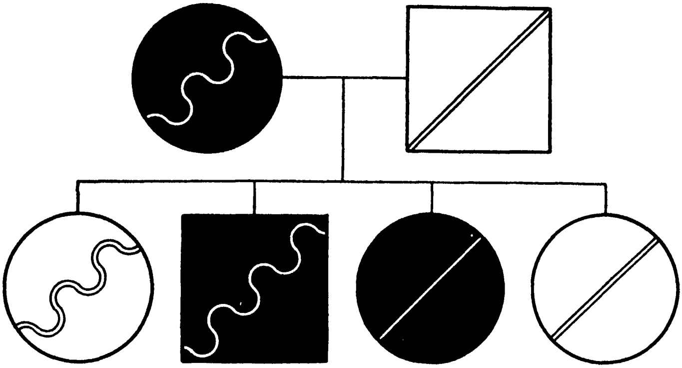


FIG. 76.—Inheritance of color and form of hair in man. Black symbols, dark hair, white symbols, light hair; wavy oblique white lines, curly hair; straight oblique white lines, straight hair. The mother must have been a double heterozygote.

F_2 Ratio Dependent on Dominance.—The squashes in the first example presented a ratio of 9:3:3:1 in the F_2 generation partly because one gene was dominant over the other in each pair. When one or both pairs of genes lack dominance, the ratio is changed. There are still 16 combinations, falling into 9 genotypes, but the phenotypes are more numerous.

What the ratio will be in such examples can be foretold by a simple calculation. Suppose that one of the pairs consists of a dominant and a recessive gene; the F_2 generation, with respect to that pair considered by itself, will be divided into two phenotypes in the ratio of 3:1. If the second pair lacks dominance, the F_2 generation with respect to it alone will consist of three phenotypes in the ratio of 1:2:1. If both pairs are studied simultaneously, the F_2 generation will present a ratio which is the product of these two, that is $(3 + 1)(1 + 2 + 1)$. Expansion of this product yields the phenotypic ratio of 3:6:3:1:2:1. This is the expected ratio in F_2 involving two pairs of genes, in one of which there is dominance, in the other no dominance.

To make this result concrete, horns and color in cattle may be used. The polled or hornless condition (P) is dominant over horns (p), but red (W) and white (w), as shown in Chap. VII, are

neither dominant nor recessive, the heterozygote being roan. Were these two pairs of characters involved in one cross, the offspring would have the genotype *PpWw* and would be polled and roan. Mating together a number of these polled roans would yield the following kinds of offspring:

Fraction	Genotype	Phenotype	Phenotypic ratio
1/16 2/16	*PPWW* *PpWW*	Polled red	3
2/16 4/16	*PPWw* *PpWw*	Polled roan	6
1/16 2/16	*PPww* *Ppww*	Polled white	3
1/16	*ppWW*	Horned red	1
2/16	*ppWw*	Horned roan	2
1/16	*ppww*	Horned white	1

Any other condition in which a heterozygote can be distinguished from both corresponding homozygotes will act in the same way to modify the F_2 ratio. For example, the blood group agglutinogens (Chap. XI) in man are both dominant; both are present in heterozygotes. Suppose that a man of blood group AB, having normal pigmentation (C) of the skin but born of an albino mother (c) so that he is heterozygous for pigmentation, marries a woman (perhaps his cousin) who is likewise of blood group AB and heterozygous for pigmentation. Their genotype is AA^BCc. While their children would scarcely be numerous enough to fall into a typical F_2 ratio, that ratio would be expected to be 3:6:3:1:2:1. The student is encouraged to verify this expectation by means of a checkerboard or otherwise and to allot the various combinations of blood group and color to the proper terms of the ratio.

If both of the pairs of genes lack dominance, or in some other way enable the heterozygote to be distinguished from the two corresponding homozygotes, so that each pair by itself

would produce an F_2 of three kinds in the ratio of 1:2:1, then the F_2 obtained from the two pairs in combination would be 1:2:1:2:4:2:1:2:1, which is derived from the product $(1 + 2 + 1)(1 + 2 + 1)$. This ratio may be made concrete by using color of flower and shape of leaf in snapdragons; for flowers which are heterozygous for red and white are pink, and plants which are heterozygous for broad leaves and narrow ones have leaves which are intermediate. It is left to the student to fit the nine different combinations of red-pink-white and broad-intermediate-narrow to the nine-term ratio just given. There is in this instance only one genotype for each phenotype, though some of the genotypes are repeated among the 16 F_2 combinations.

Combination of Sex-linked and Autosomal Character.—A special situation exists when one character which is sex-linked is tested simultaneously with another which is autosomal. The genetically well-known vinegar fly Drosophila furnishes many examples. One character involves the shape of the bristles. While the wild-type fly has bristles of a regularly and gently curved form ending in a single sharp point, a mutant type has irregularly crooked bristles often divided into a fork at the tip. These characters are sex-linked, the gene being in the X chromosome, and forked bristle is recessive. The other character chosen concerns body color. The wild-type color is usually called gray, and one of the mutants is black. The genes for these characters are in chromosome 2, hence are not related to sex. Black is recessive.

The cross is represented as between a forked gray female and a nonforked black male (Fig. 77). In the illustrations the bristle and a small portion of the body wall, enough to show the color, are given. The shorter of the two chromosome pairs is the X (or XY), the longer one is chromosome 2. The Y chromosome in Drosophila is bent and is represented as turned back at one end, as in an earlier illustration. It contains no gene that can be detected as bearing on either of the characters here used, hence it may be regarded as "empty" for the purpose of this example.

In accord with a general practice among geneticists, which may now be introduced to advantage, only the mutant genes are marked. Any chromosome not marked is assumed to have the wild-type genes in it. An unmarked X chromosome contains the nonforked gene (F), an unmarked second chromosome

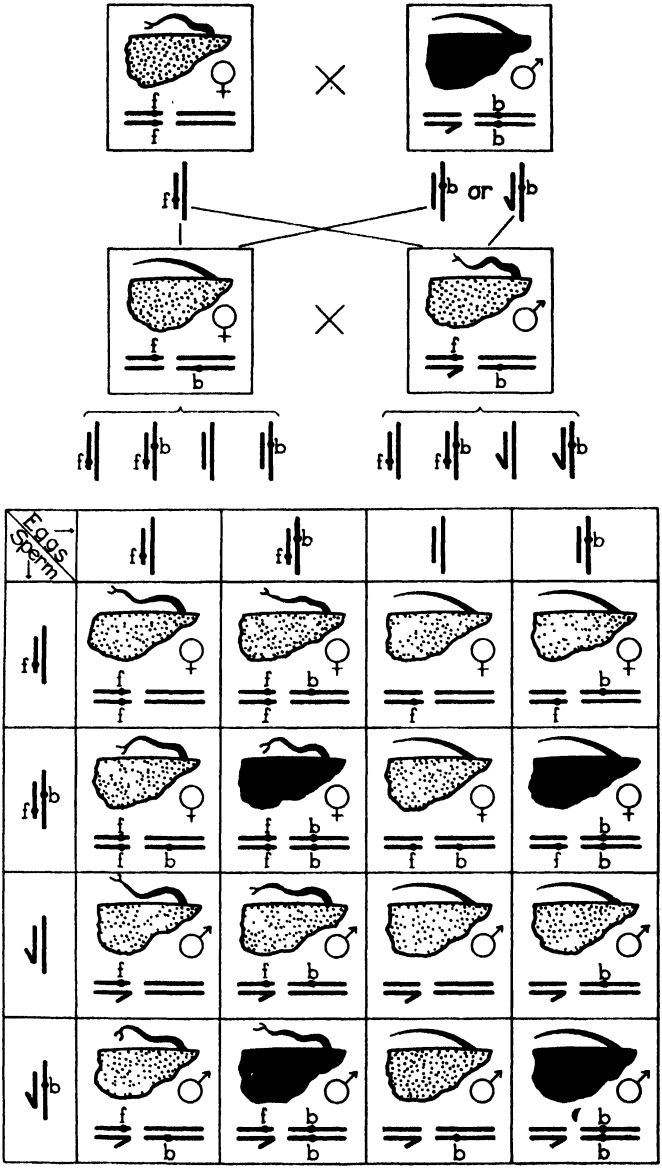


FIG. 77.—Simultaneous inheritance of a sex-linked and an autosomal character. Forked bristle is sex-linked, and recessive to single-tipped wild-type bristle. Black body is autosomal, and recessive to wild-type gray (dotted in figure). X chromosomes short, autosomes long, Y chromosome bent. *f*, gene for forked; *b*, gene for black. Chromosomes with no gene marked contain only wild-type genes; the Y chromosome, however, has no known genes relating to bristle shape.

contains the gene for gray body (B). The Y chromosome is unmarked but, as an exception, this does not imply any wild-type genes.

With these explanations Fig. 77 should be fairly clear. The germ cells are represented by vertical chromosomes, one chromosome from each pair. While the original female produces but one kind of egg, the male, because of its XY chromosomes, produces two kinds of spermatozoa, as in all instances of sex-linked inheritance in an XY species. There are consequently two combinations in F_1, one of them yielding females, the other males. Now, the males will inevitably contain the gene f, received from the mother, and will exhibit the forked bristles. The females will be nonforked, and both sexes will be nonblack (gray), because in each of them there is a wild-type (dominant) gene in one of the chromosomes of the pertinent pair.

Each sex in the F_1 generation produces four kinds of germ cells, and these are combined in 16 ways, which are shown in the illustration after the manner used in earlier figures. It is clear at a glance that the ratio of gray to black is 3:1 (12:4), as would be true of any autosomal characters. The ratio of nonforked to forked, however, is 1:1 (8:8), as would happen for any other sex-linked character involved in a cross like the one here made. When the two pairs of characters are combined, and if sex is taken into account, the F_2 generation is made up as follows:

3/16	nonforked gray	♀ ♀
1/16	nonforked black	♀ ♀
3/16	forked gray	♀ ♀
1/16	forked black	♀ ♀
3/16	nonforked gray	♂ ♂
1/16	nonforked black	♂ ♂
3/16	forked gray	♂ ♂
1/16	forked black	♂ ♂

This ratio could have been foretold at the outset by a brief calculation. The ratio in F_2 for an autosomal character (gray-black) with dominance is 3:1, with the sexes proportionately represented. The F_2 ratio for a sex-linked character, when the recessive member of the pair entered from the XX (or ZZ)

sex in the first cross, is 1:1 with the sexes in the same proportion in both classes. And finally, the ratio of the sexes in any generation is 1:1. Combining these at random results in a ratio which is obtained from the product $(3 + 1)(1 + 1)(1 + 1)$, or 3:1:3:1:3:1:3:1. A little experience enables one to assign the correct combination of characters to each term of the ratio, but the checkerboard is always available to those who feel uncertain of the algebraic method.

To the student is left the task of combining an autosomal and a sex-linked character starting with a cross reciprocal to the first mating in Fig. 77 or with any similar cross in which the sex-linked character is first introduced through the male. The F_1 generation would give no sign that a sex-linked character is involved, and the ratio in F_2 would be 3:1 for both the sex-linked and the autosomal character; but the one-fourth sex-linked recessives would be all males.

In man Fortuyn (1935), using the data of other investigators, concludes that harelip and cleft palate are due to a combination of one autosomal and one sex-linked gene. This explanation was suggested partly because of the greater prevalence of the character in males, which the reciprocal cross just mentioned shows for the autosomal-sex-linked combination.

More than Two Pairs of Genes.—Diminished importance attaches to simultaneous study of three or more pairs of characters, particularly in elementary work; yet there are situations in which it is necessary to deal with these larger combinations. The simplest such combination is one involving three pairs of genes, in each of which one gene is dominant over the other. To make the problem concrete three characters of rabbits may be used, two of them concerned with hair color, one with hair length. The two pairs of color genes are part of a more extensive scheme, and certain assumptions have to be made regarding the other members of the system, but these assumptions need not be stated here. One of the genes (G) causes the pigment on each individual hair to be broken up and limited to certain portions of the hair, making the general color gray (or agouti), the wild-type color of rabbits. The allele of this gene (g) causes the pigment to be uniformly spread along the hair, and in this example the color thus spread is assumed to be black. Nonagouti (black), as the small letter indicates, is recessive. The second pair of

genes determines how dense the pigment is, *d* making it dilute, *D* intense. Black color accompanied by *dd* becomes "blue," but with *D* it is deep black. Agouti accompanied by *dd* is light agouti, while with *D* it is the typical wild-type agouti. The third gene determines length of hair, *l* representing long hair (Angora), *L* short hair.

In whatever combination the genes enter from the parents, the genotype of F_1 is *GgDdLl*, and the phenotype is wild-type agouti and short-haired. The germ cells of F_1 are of eight kinds in each sex, equally numerous, as follows:

Eggs	Spermatozoa
GDL	*GDL*
GDl	*GDl*
GdL	*GdL*
gDL	*gDL*
Gdl	*Gdl*
gDl	*gDl*
gdL	*gdL*
gdl	*gdl*

These are combined at random in fertilization, resulting in 64 combinations among which there are many duplicates. It is advisable for the beginner to prepare a checkerboard the first time he analyzes an F_2 generation involving three pairs of characters. The diagram must contain 64 squares. The eggs may be written down the columns of squares, one kind of egg eight times in each column, and the eight kinds of spermatozoa along the horizontal rows.

Then comes the inspection of the 64 genotypes to assign them to their proper phenotypes. There are but eight of these phenotypes, for the same reason that eight kinds of germ cells are produced by the F_1 animals. These eight phenotypes are, however, very unequal in numbers as follows:

$27/64$ dark agouti short-haired
$9/64$ dark agouti Angora
$9/64$ deep black short-haired
$9/64$ light agouti short-haired
$3/64$ deep black Angora
$3/64$ light agouti Angora
$3/64$ light black short-haired
$1/64$ light black Angora

The expression "dark agouti" would ordinarily be simply "agouti," "deep black" merely "black," and "light black" only "blue"; but it seemed better in such a list to use three words, separately indicating the results of the three pairs of genes.

Short Cut to Complex F_2 Generations.—All the above could have been done without resort to a checkerboard, and with a little experience the diagram is usually discarded. The number of kinds of germ cells produced by an F_1 organism depends on the number of genes for which it is heterozygous. Since for a single heterozygous pair the number of kinds of germ cells is 2, for two heterozygous pairs the number is $2^2 = 4$, for three heterozygous pairs $2^3 = 8$, for four heterozygous pairs $2^4 = 16$, for five heterozygous pairs $2^5 = 32$ kinds, and so on. The number of combinations in F_2 is the square of the number of kinds of germ cells produced by F_1, that is, $2^2 = 4$, $4^2 = 16$, $8^2 = 64$, $16^2 = 256$, and $32^2 = 1024$ for the respective numbers of heterozygous pairs of genes just named. The number of phenotypes into which these F_2 combinations fall is the same as the number of germ cells produced by F_1, provided one gene of each pair is dominant. Four F_2 combinations are divided between 2 phenotypes, 16 F_2 combinations among 4 phenotypes, 64 F_2 combinations fall into 8 phenotypes, 256 into 16, and 1024 into 32. All this is based on the assumption that one gene is dominant over the other in each pair.

The numerical ratio of the various phenotypes is a power of the binomial $3 + 1$ whose exponent is the number of pairs of heterozygous genes in F_1. For 2 heterozygous pairs it is $(3 + 1)^2 = 9 + 3 + 3 + 1$. For 3 heterozygous pairs it is $(3 + 1)^3 = 27 + 9 + 9 + 9 + 3 + 3 + 3 + 1$. For 4 heterozygous pairs it is $(3 + 1)^4 = 81 + 27 + 27 + 27 + 27 + 9 + 9 + 9 + 9 + 9 + 9 + 3 + 3 + 3 + 3 + 1$. Again it is assumed that there is dominance in each pair. Longer expansions may be omitted.

How the several phenotypes are allotted to the various numbers in these ratios may be illustrated concretely from the rabbit example described in the preceding section. It will be observed that the most numerous class, which makes up $^{27}/_{64}$ of the whole F_2, consists of those individuals exhibiting the dominant character of each pair. Each of the $^{9}/_{64}$ classes exhibits two dominants and one recessive, but there are three combinations that

meet this specification. Each of the $\frac{3}{64}$ classes shows one dominant and two recessive characters, while the $\frac{1}{64}$ class shows all of the recessives.

When these relations have been mastered, it is possible to write down the phenotypic composition of even a very complex F_2 generation without using a checkerboard or any comparable aid.

CHAPTER XIV

INTERACTIONS OF GENES

The two kinds of independence of genes referred to in the preceding chapter have no relation to each other. It often happens that genes which are in different pairs of chromosomes and are therefore independent in their distribution to the germ cells pool their activities when it comes to producing their characters. It is this type of interdependence with which the present chapter deals. Something that might be called interaction of genes we are already familiar with as between different genes at the same locus. Dominance is at stake in such interactions. Though one gene, when they are not alike, may succeed in prevailing over the other and so produce its character which is then said to be dominant, very frequently the combination of two different genes of the same pair may lead to an intermediate expression, or at least to a character unlike that which would arise from two similar genes of either of the kinds. Mutant members of multiple allelic series commonly bear this relation to each other, as stated in Chap. XI. This lack of dominance is to be regarded as due to interaction between the alleles. This chapter, however, deals only with pairs of genes at different loci—usually, indeed, in different chromosomes.

Interaction between Dominant Genes.—Some breeds of fowls have what is called the pea comb (Fig. 78, *A*), which is due to a dominant mutant gene (P) at a certain locus. Without this mutation (hence with p) the comb is of the high, thin, notched form which approaches the presumable wild-type comb of ancestral birds and which is called single. Crossing pea with single yields pea in F_1, and pea and single in the ratio of 3:1 in F_2. In another chromosome there is, in certain varieties, a dominant mutant gene (R) which results in rose comb (Fig. 78, *B*). Without this mutation (hence with r) the comb is likewise single, and crossing rose with single gives rose in F_1 and rose and single (3:1) in F_2. At least two pairs of genes at

different loci must remain as they were in the ancestral fowls in order that the comb may be single. Change either one of them to a dominant gene and the comb becomes something else. Now, what will happen if genes at both loci are changed to the dominant ones at the same time? The answer may be learned by crossing the two kinds of fowls. The pea-combed bird has the genotype $PPrr$, the rose-combed fowl $ppRR$. Their hybrid must be $PpRr$. Very unexpectedly (to one who has not

FIG. 78.—A character due to interaction of genes. Walnut comb (C) is produced when the dominant genes for pea comb (A) and rose comb (B) are both present in the same fowl.

made the test) its comb is like that in Fig. 78, C, which is called walnut. This new comb is the product of the combined action of P and R.

The F_2 generation will consist of a $9/16$ class possessing at least one P and at least one R—and having the phenotype briefly designated PR[1]—whose combs are walnut; a $3/16$ class of phenotype Pr which has pea combs; another $3/16$ class of phenotype pR which is rose-combed; and a $1/16$ group phenotypically pr which has single combs. The ratio is not at all disturbed;

[1] This common practice among geneticists should not prove confusing. When referring to the phenotype, a single symbol for each pair of characters designates the character and is not the formula of a germ cell, as a single symbol would otherwise be.

there is merely an interaction between the two dominant genes affecting the comb such that a totally different form of comb is produced. How the two genes together modify physiological processes so that comb development is changed is not known.

Color in sweet peas also results from interaction between two dominant genes. To have colored flowers, a plant must have what is called a *chromogen* (a basic substance out of which colored matter may be developed) and an *enzyme* which will convert the chromogen. Both the chromogen and the enzyme are colorless, but the two together produce color in the flowers. The genes for these substances have commonly been designated *C* and *R* respectively. A sweet pea with the genotype *CCrr* is white because it forms only the chromogen. A pea whose genotype is *ccRR* is likewise white, for it has only the enzyme. When they are crossed, however, the F_1 plants are of the genotype *CcRr*, both chromogen and enzyme are produced, and the flowers are colored. When these colored F_1 plants are self-fertilized, the F_2 generation consists of a $9/16$ class whose phenotype would be indicated by CR, which is colored; a $3/16$ class Cr which has white flowers; a $3/16$ class which is cR and therefore white; and a $1/16$ class which is the doubly recessive cr, likewise white.

Here the ratio of the F_2 phenotypes is changed to 9:7, for the last three classes of the 9:3:3:1 distribution are quite indistinguishable. Such modifications of expected ratios are quite common where genes interact. To get a 9:7 ratio the interacting genes must be such that neither one produces anything visible by itself, but the two together yield a perceptible product.

There must be many such interactions between dominant genes in man, though it is difficult to discover them with certainty from family histories. Lenz (1936) regards the disposition to stomach ulcers as a result of such a combination. These ulcers are in part, of course, due to environment. The hereditary component appears to be partly allergy, or hypersensitiveness to certain substances, partly irritability of the vagus nerve; and these are in general dominant characters. If longevity be regarded as one quality, as it sometimes is, it must be a resultant of numerous genes, both dominant and recessive, working together. Length of life flows from the combined influences of health, disease, defects, strengths, and weaknesses, in whose

genetic bases, as will be seen later, both dominant and recessive genes are liberally represented.

Interaction between Recessive Genes.—In summer squashes, as already indicated (Fig. 74), disk-shaped fruit (D) is dominant over spherical (d). Now, there is another pair of genes also concerned with shape. One gene at this other locus makes the fruit disk-shaped, is dominant, and may be symbolized by F (from flat). Its allele (f) makes the fruit spherical. There are thus two kinds of spherical squashes, one genotypically *ddFF*, the other *DDff*. Being homozygous for either d or f makes the fruit spherical; but to be disk-shaped a fruit must contain both D and F.

If the two spherical types are crossed, their hybrid is *DdFf*, and it is disk-shaped. If the F_1 is self-fertilized, the F_2 generation consists of a $9/16$ class phenotypically DF which is disk-shaped; a $3/16$ class Df which is spherical; and a $3/16$ class dF which is likewise spherical. Finally, the $1/16$ class df, perhaps not surprisingly, has elongated fruit.

There are two interactions here. Since disk shape requires both D and F, these genes must cooperate to produce that form. This is comparable to the interaction which produces walnut comb or colored sweet peas, since it occurs between two dominant genes. The second interaction is between the two recessive genes d and f, to produce elongate fruit. In this joint action, one gene may be regarded as merely accentuating the effect of the other. For, taking disk fruit as the standard of comparison, spherical fruit itself is somewhat elongate. The two genes for spherical form merely increase this elongation.

The F_2 ratio in this cross is 9:6:1. To yield this ratio, the two genes, taken singly, must produce similar characters, but the two dominants together or the two recessives together must produce something different.

It is difficult to prove specific interactions in man, but there is little question that they occur in abundance. A scheme of inheritance of human hair color adopted by Lenz (1936) involves several of them. As an example, the color he designates blond rests first of all on a recessive gene for general pigmentation of medium degree and next upon another recessive gene responsible for a lack of melanization (black pigment formation). There are other genes, and the whole system is described in a later

chapter. The part here indicated illustrates interaction between recessive genes. The distinction between dominant and recessive in such interactions is not, however, important.

Modifiers.—When a gene produces no observable effect in one genetic background, but changes the character produced by another gene when that gene is present, it is called a *modifier.* One modifying gene was introduced into the preceding chapter without calling attention to its nature. That gene is the diluting factor in rabbit color which changes black to "blue" and agouti

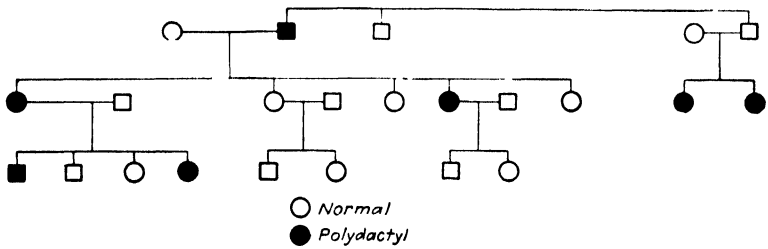
FIG. 79.—Polydactyly in both hands and feet. Thumbs and great toes are either duplicated or branched. (*From Atwood and Pond in Journal of Heredity.*)

to light agouti. The diluting gene can be detected only in the presence of a color gene or combination.

Agouti itself is a modifier, for its effect is to rearrange the color (black or brown) produced by other genes. In the absence of the agouti gene the pigment is spread more or less uniformly along the hairs. With the agouti gene, the pigment is restricted by yellow. In the absence of the genes requisite to color of any kind (in an albino animal), the agouti gene cannot be expressed.

Sometimes the dominance of one gene is modified by other genes. The bristle character known as forked in Drosophila (Fig. 77) is ordinarily recessive; but in the presence of a certain

other gene, which has no other known effect, forked shows to some extent even in a heterozygote. It is not at all unlikely that extra fingers in man (polydactyly, Fig. 79) has its dominance determined in a similar manner. It is well known from family histories that extra fingers may show in a heterozygote, or they may be missing in a heterozygote. Figure 80 presents evidence of this capriciousness. When present, the extra fingers may be



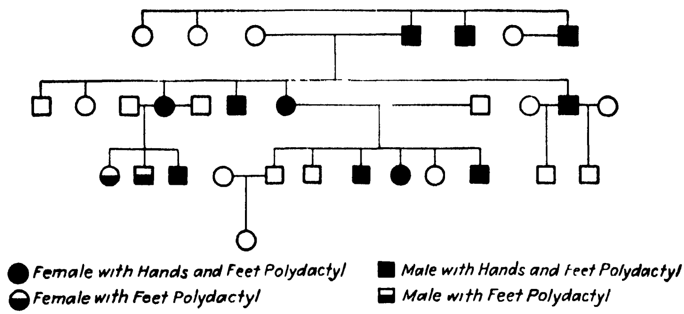


FIG. 80.—Two family histories of polydactyly which, together, indicate that the character is sometimes recessive, sometimes dominant. (*From Milles in Journal of Heredity.*)

well developed or quite small (Fig. 81, contrasted with Fig. 79). This variable condition has long been known as "irregular dominance." From what is known of other animals, it seems probable that this variable expression of extra fingers is due, not to any variability of the polydactyly gene itself, but to certain other genes which may or may not accompany it. It has been impossible to identify any such genes, or to trace them through lines of descent, in man. If they exist, they too are modifiers.

Spotting factors limit the distribution of colors in various organisms. Colored beans homozygous for a spotting gene are mot-

tled, and colored mice homozygous for a spotting gene have white areas of variable size and shape along with colored ones. Mice without any color gene (albinos) cannot, of course, exhibit spots.

So many examples of modifying genes have been discovered that it seems likely that every gene is thus related to others. Probably every gene has its effect partly determined by other genes, and in turn is a modifier of other genes. Characters are thus produced not by certain genes but by the whole genetic complex or a considerable part of it.

Dominance Modified by Sex.—Just as dominance of forked bristles in Drosophila and perhaps of polydactyly in man is

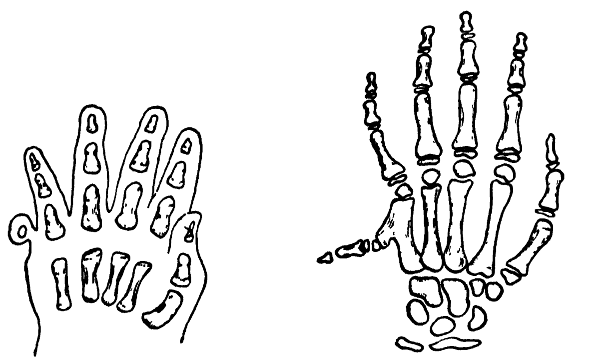


Fig. 81.—Skeletons of polydactyl hands illustrating, respectively, slight and intermediate development of extra fingers. Strong development is shown in Fig. 79. *(From Milles in Journal of Heredity.)*

influenced by other genes, so is dominance of certain characters dependent on sex. These characters are dominant in one sex, recessive in the other. Horns in sheep behave in this way, at least in certain breeds. In one breed both sexes are horned, though the horns of rams are larger than those of ewes. In another breed both sexes are hornless. When these breeds are crossed, the male F_1 offspring are horned, while the females are hornless. The character is not sex-linked, as one might first suppose on obtaining such a result, for both reciprocal crosses between the breeds yield the same distinction between the sexes. That is, in the first cross the horns may be introduced either through the female or through the male; in either case the male progeny are horned, the female hornless. Were horns sex-linked, only one of the two reciprocal crosses would yield an F_1 in which the sexes were different.

When an F_2 generation is obtained from these F_1 sheep, there are some horned and some hornless in each sex; but among the males the ratio is 3 horned to 1 hornless, while among the females it is 1 horned to 3 hornless. All of these peculiarities are explained on the assumption that a heterozygote (*Hh*) has horns if it is a male, no horns if it is a female. One homozygote (*HH*) is horned regardless of sex, the other homozygote (*hh*) is hornless in either sex.

Baldness in man is held by some investigators to be inherited in the same sex-influenced ways; that is, it is dominant in men, recessive in women. It is true that many more men than women are bald, but it is doubtful whether the sex influence is precisely as stated. The gene for baldness must be very common to explain the number of bald men, and, with this prevalence in the population, there should be quite a few homozygous (hence bald) women. Doubtless there are more bald women than is generally appreciated but hardly as many as there should be if all homozygous women were bald. A plausible suggestion is that baldness may sometimes be suppressed, or nearly so, even in homozygous women. If sex can prevent *one* gene from producing its character, it is not unlikely that it might prevent even a pair of them from coming to expression. This suggestion is the more reasonable because just such suppression of a character occurs in the male of the clover butterfly. This species of butterfly is typically yellow, but it has a white variety. The white color is exhibited by females which have the gene for it, but never by the males. Even homozygous "white" males are not white. Males transmit the white gene if they possess it (transmit it to all their offspring if homozygous), and their daughters may be white, but their sons are only yellow. Something in the male physiology prevents the white genes from producing any visible effect even when there are two of them. If the female physiology in man has this effect upon baldness, at least partially, the number of bald women could be far below expectation. Probably one or more of the female hormones should be held responsible for whatever suppression or reversal of dominance there is.

Epistasis.—When two genes occupying different loci (hence not allelic to each other) affect the same feature of an organism, they often bear the relation, one to the other, known as *epistasis*.

This term has been used in two senses. Sometimes it is used merely to mean that one gene depends on the presence of another gene to produce any effect. The diluting gene already referred to in connection with hair color is an example. It has no effect unless a color gene accompanies it. Modifiers in general have no effect unless the gene whose action they are capable of changing goes with them. Mammals in general must have a fundamental color gene *C* in order to be pigmented at all. What color they will exhibit is then determined by one or more other genes. Some geneticists have used the word epistasis to indicate the relation of the overlying genes in all these examples to the genes on which they are dependent. The genes of a mouse which determine the nature of the color are all, in this sense, epistatic to the basic gene *C*. Modifiers are epistatic to the genes they influence.

Another and somewhat cruder but simpler form of epistasis is the concealment of one character by another. According to one proposed scheme of hair color in man, there is a gene for red pigment and another for black or brown pigment. When both genes are present, both pigments are produced; but if the black pigment is heavy the red may be completely hidden. Black is then said to be epistatic to red.

Inhibitors.—A special form of epistasis is shown by inhibiting genes. One gene may entirely prevent another (nonallelic) gene from producing its character. The absence of color in White Leghorn fowls is due to an inhibitor. Such a fowl may have genes which would make it black, or brown, or barred; but, if the inhibiting gene (*I*) is present, the color genes are entirely inoperative and the plumage is white. The correctness of this explanation is attested by hybrid generations in which, due to the shuffling and recombination of the genes, some fowls receive the color genes and not the inhibitor, and consequently they are colored.

Sheep may also possess a dominant inhibiting gene which prevents black color of their wool. Such sheep are white but are capable of transmitting black color to some of their progeny in which the inhibitor may be absent.

White color in squashes is a further example of inhibitors. The inheritance of white as contrasted with yellow has already been described in Chap. XIII, Fig. 74. The story is not quite

so simple as there told, however, for squashes may have a third color, green. When yellow is crossed with green, yellow proves to be dominant over green, even though it is recessive to white. There must be a second pair of genes *Yy*, which determines whether the squash can be yellow or not. A squash of genotype *wwYy* is yellow, while *wwyy* is green. The inhibiting gene in squashes is *W*; when this is present, as in *WwYY* or *WWyy*, neither the yellow nor the green color can be produced, and the squash is white.

It has been suggested that there may be in some human beings a gene inhibiting brown pigment in the iris of the eye. This suggestion is made to explain the occasional instances of brown-eyed children derived from parents both of whom are blue-eyed. As has been indicated (Fig. 43), absence of brown pigment is recessive to its presence, that is, blue is recessive to brown. Under these circumstances all blue-eyed persons should be homozygous, and two blue-eyed parents should have only blue-eyed children. The occurrence of a brown-eyed child in the family of two blue-eyed parents is thus exceptional and requires an explanation. One possibility is that one of the parents is blue-eyed because of an inhibitor. That parent is supposed to have a gene for brown pigment, but also a dominant inhibiting gene which prevents the pigment from developing. If such parent is heterozygous for the inhibitor (*IiBB* or *IiBb*), some of the offspring may receive the gene *B* but not *I*. These offspring will be brown-eyed.

Complex Interaction Groups.—Most interactions of genes probably involve a number of loci, even if only two have been discovered. Some rather complicated systems of interaction have been thoroughly established. One of the simplest schemes of multiple-gene interaction is that in which the genes at the several loci have similar effects and in which their product is cumulative. An example is red color in the grains of wheat, as contrasted with white grain. The red color is due to three pairs of genes, all having the same effect and perhaps, though not necessarily, equal effects. It is common practice among geneticists to symbolize such similar genes by subscripts to a common base—R_1, R_2, and R_3. None of these genes is dominant over its allele (r_1, r_2, r_3) which produces no red pigment, so that heterozygotes are intermediate. Thus, $r_1r_1r_2r_2r_3r_3$ is white,

$R_1r_1r_2r_2r_3r_3$ has a little red color, and $R_1R_1r_2r_2r_3r_3$ has somewhat more red. Each pair behaves in the same way, so that $r_1r_1R_2R_2r_3r_3$ is a little redder than $r_1r_1R_2r_2r_3r_3$. Now, the effects of the genes at different loci are cumulative. This means that $R_1r_1r_2r_2R_3r_3$ is darker red than $R_1r_1r_2r_2r_3r_3$, and perhaps of about the same color as $r_1r_1r_2r_2R_3R_3$ or $r_1r_1R_2R_2r_3r_3$. Any two genes, whether at the same locus or at different loci, produce more red than one, three produce more than two, four more than three, and so on. If it be assumed, as is approximately if not wholly true, that the genes at the three loci have equal effects, then three genes produce a certain amount of red color no matter which of the six places they occupy. Four genes, in any of the positions, would produce a deeper red, five genes still deeper, while six would duplicate the original red variety, which is $R_1R_1R_2R_2R_3R_3$. A hybrid generation (F_2 or later) could thus include six different grades of red in addition to strictly white wheat.

More complicated interactions occur when the genes at different loci have qualitatively different effects. Coat color in mice will serve as an illustration. With the omission of the yellow color, which has already been presented as lethal in homozygotes (Fig. 68), the system is in part as follows. To be colored at all, a mouse must have the basic dominant gene *C*; if its genotype is *cc* it is albino. If in addition to *C* the mouse has the gene *A*, the pigment in the hairs is restricted to certain parts and the coat is agouti. In the absence of *A*, that is, in *CCaa*, the pigment is spread along the entire hairs and solid color results. What that color is depends on still another pair of genes. If that pair includes *B*, the mouse is solid black, while *bb* is brown. The effect of the agouti gene *A* is the same for black and brown mice; but in mice with *B* it produces the typical wild agouti coat, while with *bb* the banding of the color on the individual hair results in the color called cinnamon. Another pair of genes determines whether the pigment granules are clumped or are spread evenly. The clumped condition results in a dilute color (*d*) as contrasted with the dense color (*D*) due to even distribution. Dilution affects any of the colors produced by the other genes, so that there are dilute agouti, dilute black (blue), and dilute brown mice. Still another pair of genes (*Pp*) determines the amount of pigment. The mutant member (*p*) of this pair causes the iris of

the eye to be reddish, owing to reduction of pigment, and is known as the pink-eye gene. Its effect on the hair is likewise to reduce the amount of pigment, giving the mouse a faded appearance. Any of the colors may be washed out by this pink-eye reduction, just as they may be diluted by the clumping of the granules (*d*). Finally, distribution of pigment over the body is governed by a sixth pair of genes (*Ss*). In *ss* individuals the pigment is restricted to spots, and mice of any color may be spotted.

A wild-type mouse must have the dominant gene of each pair, hence if true-breeding (homozygous) must have the genotype *CCAABBDDPPSS*. Substitute for any one of these pairs their recessive mutant alleles, and a different kind of mouse is produced. As intimated above, even this is not the complete known scheme of coat color, and it is likely that some features of it remain to be discovered as new mutations arise.

Human hair color is probably as complicated as that of the smaller mammals, but conjectures concerning that system are reserved for a later chapter, where a number of hereditary traits in man are assembled for reference.

Blending Characters.—The color of wheat, described in the preceding section, exemplifies what are known as blending characters. This name was applied to them because they did not *appear* to show the sharp segregation which Mendelian heredity ordinarily involves. The distinctive marks of a blending character are for the F_1 generation to be intermediate between the parents (and fairly uniform among themselves if the parents were homozygous) and for the F_2 generation to break up into a variety of types ranging, by small steps, practically from one grandparental extreme to the other. These conditions are met, it will be observed, in red and white wheat. When these two varieties are crossed, according to the scheme presented, each F_1 plant will contain three of the six red genes ($R_1r_1R_2r_2R_3r_3$), and will be about half as red as the red variety. Since all F_1 plants have the same genotype, that generation has nearly uniform color. In the F_2 generation, however, a plant may have any number of red genes from none at all to six. This generation is accordingly very variable in color. A small fraction of the plants should be fully red and an equal portion strictly white, while the bulk of them would be intermediate in various degrees.

It is very common for quantitative characters, such as length, weight, or depth of color to be inherited according to the blending scheme. The probable explanation in most such instances is that many pairs of genes having somewhat similar effects are at work. There are other possibilities, such as multiple alleles, and one such possibility for human hair color is described in a later chapter.

CHAPTER XV

MODIFIED F_2 RATIOS

While the examples used in the preceding chapter are sufficient to illustrate the principle of interaction among genes, a few other instances of intergene influence should be presented because of their effect on F_2 ratios. These ratios of the different classes of individuals are used by the experimenter to arrive at a tentative judgment of the genetic situation being investigated. A ratio of 3:1 or 1:2:1 in F_2 tells him at once that probably only one pair of genes is involved. When that generation includes four classes or when there are three classes in some other ratio than 1:2:1, it is assumed that at least two pairs of genes are concerned. It is important therefore that the geneticist know the more common ratios which may appear in F_2 and the explanation of them.

The last chapter presented two modified ratios. One was 9:7, which is expected from two pairs of genes if neither dominant produces anything visible by itself, but the two in unison do yield something perceptible. It was exemplified by color in sweet peas, after a cross between two white varieties. The other ratio illustrated was 9:6:1. This distribution is expected when two pairs of genes at different loci have similar effects when they are alone, a different effect when both are together in one individual, and a still different effect if both are absent. The two genes for spherical shape in squashes, each capable of producing that form in the absence of the other, cooperate to produce the elongate shape when they are together and the disk shape if both are missing. The principal other modifications of F_2 ratios are given in the following sections.

The Ratio 9:3:4.—The complex scheme of color inheritance in mice offers an opportunity to obtain a ratio of 9:3:4 in F_2. To be colored at all, a mouse must possess the gene C. If it has only cc at that locus, it is albino, even though a number of other genes for particular colors or distribution of color may

be present. To use only two of the genes belonging to this system, assume that a mouse is of the genotype *CCaa*. The gene *a* is the nonagouti gene, which means that the mouse will be of solid color—say, black. Another mouse, of genotype *ccAA*, will be albino, even though it has a gene (*A*) which would make it agouti if it had color of any kind. If these two mice are crossed, their offspring are *CcAa*, hence agouti. Mating these F_1 mice among themselves yields the following F_2 generation:

Fraction	Genotype	Phenotype	Ratio
1/16	*CCAA*	Agouti	9
2/16	*CcAA*		
2/16	*CCAa*		
4/16	*CcAa*		
1/16	*CCaa*	Black	3
2/16	*Ccaa*		
1/16	*ccAA*	Albino	4
2/16	*ccAa*		
1/16	*ccaa*	Albino	

The 9:3:3:1 ratio, expected from two pairs of genes, is converted into 9:3:4 because the last two classes are phenotypically identical. This ratio regularly occurs when, of two interacting dominant genes, one has a detectable effect by itself, while the other has none. The gene *C* (under the assumptions made for this illustration) has an effect by itself, *A* does not.

The Ratio 12:3:1.—In oats there is a variety with black chaff, another with white. When these were crossed, the offspring had black chaff, and there was every expectation that the original black and white varieties would prove to differ in only one pair of genes and that the F_2 generation would divide between the black and the white type in the ratio of 3:1. To the breeder's surprise, however, while there were blacks and whites in F_2, there was also a third class with gray chaff, and this kind was several times as abundant as the white.

The explanation was that the black variety had, in addition to the gene *B* for heavy black pigment, also the gene *G* for a smaller amount of black pigment, which by itself would make the

chaff gray. The black oats was thus of the genotype *BBGG*. Either the effect of *G* was completely concealed by that of *B* or the two genes cooperated to produce a result identical with that of *B* alone. The white-chaffed oats had the recessive genes of both pairs (*bbgg*), hence had no pigment.

The F_1 generation was *BbGg*, and was black. The F_2 generation consisted of $\frac{9}{16}$ BG (using the customary way of indicating characters by just one symbol), $\frac{3}{16}$ Bg, $\frac{3}{16}$ bG, and $\frac{1}{16}$ bg. The first two of these classes were indistinguishable, black; the third was gray; the fourth was white. The phenotypic ratio is thus 12 black to 3 gray to 1 white. It is derived from the 9:3:3:1 ratio by combining the first two terms.

The 12:3:1 ratio is obtained from two pairs of genes when each dominant gene has a visible effect by itself, but one of them conceals the other or the two cooperate to produce an effect identical with that of one of the genes alone.

The 13:3 Ratio.—As has already been noted in an earlier chapter, the white plumage of White Leghorn fowls is nearly dominant over the colors black, brown, or barred. Since white plumage in some other breeds, the Wyandottes and Plymouth Rocks for example, is recessive to color, the dominance of the Leghorn white is peculiar. The reason for the difference is that the white of Leghorns is due to an inhibiting gene (*I*) which prevents the color genes, if any, from coming to expression, whereas the white of Wyandottes and Plymouth Rocks is due to the absence of genes producing color.

When the breeds are crossed, these two kinds of white may be brought together. The White Leghorn may be indicated by *IICC*, the gene *C* being that for color, which is inhibited by *I*. The white Wyandotte is *iicc*, since it lacks both the inhibitor and the basic color gene. The F_1 fowls are *IiCc*, hence white. The F_2 derived from them are $\frac{9}{16}$ IC, $\frac{3}{16}$ Ic, $\frac{3}{16}$ iC, and $\frac{1}{16}$ ic. Of these classes, three (IC, Ic, and ic) are white (the first because of the inhibitor, the third because no color gene is present), while one (iC) is colored. The ratio in F_2 is thus 13 white to 3 colored.

Inhibiting genes are the primary cause of this particular modification of the 9:3:3:1 ratio.

Duplicate Genes.—Sometimes a character may be produced by either one of two pairs of genes at different loci. These genes are identical. If they are not cumulative in their effect, both

pairs may exist in the same individual and still produce only the character which either one alone would determine. Such genes are known as *duplicate* genes. They do not afford an example of interaction of genes, but they do cause a striking modification of the expected F_2 ratio.

One of the earliest known instances of duplicate genes concerned shape of seed capsule in the weed shepherd's-purse. Among the several forms which the seed capsule may assume are the common triangular one and the spindle shape. When plants with triangular capsules were crossed with those having spindle-shaped capsules, the F_1 was triangular. The F_2, as was expected, divided between the two forms; but the triangular ones were much more abundant than three-fourths of the total.

The reason for this unexpectedly large proportion of triangular capsules is that two pairs of genes, identical with each other, produce that shape. These genes have been called C and D. The triangular-capsuled plants were $CCDD$, the spindle-shaped $ccdd$. Their hybrid ($CcDd$) had triangular capsules. When these double heterozygotes were self-fertilized, the 16 F_2 combinations were those shown in Fig. 82. The formulas there shown are separated into their egg and pollen components but may easily be recast to bring the two genes of the same pair together as is usually done. Any of these F_2 plants which have at least one C or at least one D will have triangular capsules. Having both C and D does not alter the shape, the capsule is still triangular. Fifteen of the sixteen combinations thus yield triangular capsules, only one spindle-shaped. This 15:1 ratio is derived from 9:3:3:1 by combining the first three classes. Duplicate genes without cumulative effect are regularly expected to produce this modified ratio.

That duplicate genes are the true explanation of the capsule shapes of shepherd's-purse is readily shown by breeding the F_2 plants further. If the various plants of that generation be self-fertilized, they produce one or both kinds of offspring, depending on their genotypes. The ratios placed below the illustrations in Fig. 82 indicate the proportion of triangular to spindle-shaped in the progeny of the respective plants. The ratio 1:0 means that all plants are triangular, while 0:1 indicates that all are spindle-shaped. The other ratios involve both types. Any plant which is homozygous for the recessive genes of one pair and heterozygous for the other pair yields triangular and spindle-

shaped in the ratio of 3:1; while a plant heterozygous for both pairs produces the same types in the ratio of 15:1. Finding that certain proportions of the triangular F_2 plants yield these several ratios (1:0, 3:1, and 15:1) is evidence that duplicate genes are the correct explanation.

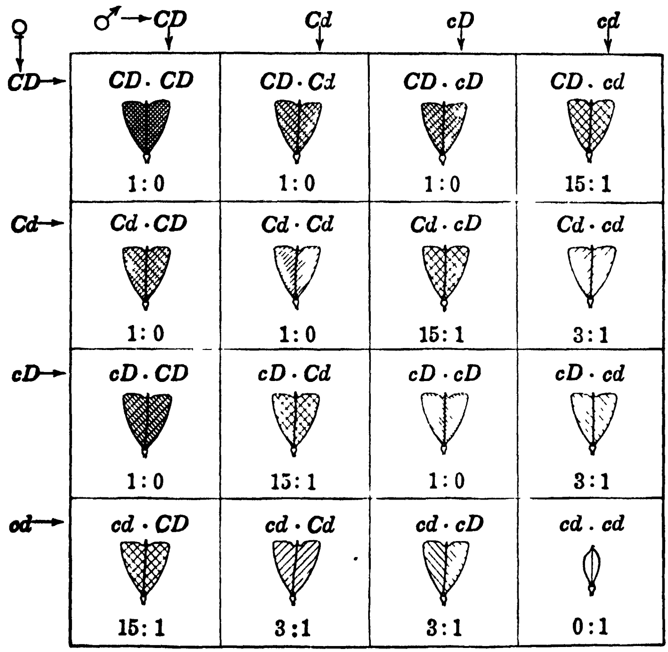


FIG. 82.—Duplicate genes in the inheritance of the shape of seed capsule of shepherd's-purse. The formulas are those of the F_2 generation from a cross between a variety with triangular seed capsule and a variety with spindle-shaped seed capsule. The factors C and D both produce triangular capsules, c and d spindle-shaped ones. C and D together produce exactly what either one alone would produce. The result is a 15:1 ratio of the two kinds of plants. This is the same as the 9:3:3:1 ratio with the first three classes visibly alike. If the plants of the F_2 generation are self-fertilized, they produce triangular and spindle-shaped offspring in the ratios indicated in the respective squares. (*From G. H. Shull.*)

The origin of duplicate genes is almost certainly to be found in the doubling of the chromosomes. The species of shepherd's-purse used in the foregoing experiments has 32 chromosomes per cell, but there is evidence that it came from a species having 16 chromosomes, merely by duplicating each chromosome. If there was one pair of genes for capsule shape in the 16-chromosome species, there would be two pairs in the 32-chromosome species. The two pairs would be identical in nature. They

would commonly be designated by the same letter with different subscripts (T_1t_1 and T_2t_2, respectively), but the earlier designations C and D have here been preserved to correspond to the illustration.

Three Pairs of Genes.—Modified ratios are as much to be expected when three pairs of genes interact as when there are only two pairs. One example will suffice to illustrate what happens. Color in the flowers of sweet peas has already been used to show the effect of interaction between two genes. One of the genes (C) produces a chromogen, the second (R) an enzyme which converts the chromogen into a colored substance. To these may now be added a third gene B, a bluing factor. If C and R are together in a given plant, the flower is red. If in addition the plant has the gene B, the red is converted into purple.

Suppose that a plant with the genotype *CCRRbb* (which is red) is crossed with one which is *ccrrBB* (which is white). The offspring are *CcRrBb*, and are purple. When these F_1 plants are self-fertilized, they produce the kinds of F_2 shown in the following table, in which only the genes coming to expression are shown:

Fraction	Expressed genes	Phenotype	Ratio
27/64	*CRB*	purple	27
9/64	*CRb*	red	9
9/64	*CrB*	white	28
9/64	*cRB*	white	
3/64	*Crb*	white	
3/64	*cRb*	white	
3/64	*crB*	white	
1/64	*crb*	white	

The usual trihybrid ratio, which is indicated in the first column of the above table, is converted into 27:9:28 by combination of its last six terms. Some of the white plants are white because they lack the enzyme, some because they lack the chromogen, some because both chromogen and enzyme are missing.

CHAPTER XVI

MODIFICATION BY ENVIRONMENT

The development of an animal or plant rests on an interplay of forces of various kinds. Since genes may modify the action of other genes in this joint operation, as illustrated in the last two chapters, it is not surprising that environment should also have an influence. The accomplishments of genes and other internal factors are brought about through physical and chemical properties. Now, the environment includes comparable agencies. Wherever the external world is brought into contact with the inner workings of an organism, there is at least a possibility that it may change the ordinary developmental processes. Of especial interest to the student of heredity are those influences which modify some otherwise well-defined genetic result.

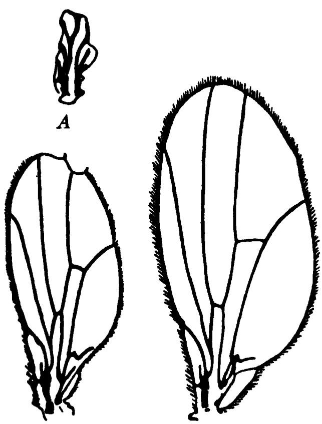
Influence of Temperature.—Among the numerous color varieties in the Chinese primula are one with red and one with white flowers. Under ordinary circumstances these two types are quite distinct, each one breeding true. In greenhouses, where temperatures are apt to range from 13° to 18°C., or even in dwelling houses with temperatures of 20° to 22°C., the distinction is maintained. But if a plant of the red variety is grown at a temperature of 35°C., its flowers are white. If a plant of the red type is raised at 18°C. until it begins to blossom, its flowers being red, and then is transferred to 35°C., the flowers that open the next few days will be red, but those opening later will be white. At the higher temperature flowers continue to open white; but if the plant is returned to the lower temperature, after a few days the new flowers open red as before. The effect of high temperature is strictly temporary. The genes distinguishing the two varieties have not been changed.

In the vinegar fly Drosophila the eye is regularly large and elliptical; but a genetic modification results in the Bar eye (Fig. 65), in which the separate elements or ommatidia are limited to an irregular vertical band. The size of this band

depends on the temperature at which the flies are raised, the higher the temperature, the smaller the band. At low enough temperatures Bar eye is almost like the wild-type eye.

In Drosophila also is a mutant variety in which the legs are branched or reduplicated. Flies of the reduplicated stock all have the gene for this peculiarity, but, to show the characteristic, they must be raised at a low temperature. The mutation was discovered when a stock of flies had been inadvertently allowed to grow in a cold room.

A certain albino variety of barley lacks chlorophyll if the temperature at which it is raised is below 7° or 8°C. This failure is due to a mutant gene. If, however, it is kept above 18°C., chlorophyll develops, and the plants are green.



A B C

FIG. 83.—Effect of temperature on vestigial wing in Drosophila. *A*, vestigial at normal temperature; *B*, at high temperature; *C*, wild-type wing. (*From Li and Tsui in Genetics.*)

Vestigial wing in the vinegar fly, a short and crumpled wing which is held out from the body and is useless for flight, is subject to change by temperature (Li and Tsui 1936). High temperature makes the wings more nearly normal, but it must be applied to the larva; keeping the pupa warm has no such effect. The vestigial wing developed at ordinary temperature and that at high temperature are shown with the wild-type wing in Fig. 83.

In all of the foregoing examples the environmental effect is observed only in individuals having a certain gene. Primulas must have the red gene, Drosophila the duplication (page 116) for Bar eye or the gene for branched legs, barley the gene for albinism, in order that temperature may have the effect indicated. Each of these varieties differs from the normal organisms by just the one pair of genes.

Temperature influences of a somewhat different order are obtained in butterflies by subjecting the pupa to extremely low temperature. These pupae would ordinarily pass the winter exposed to whatever temperatures prevail in their respective geographic regions. If kept much colder than that, the butter-

flies emerging from them in the spring often have a different color or pattern from the untreated individuals. Occasionally this excessively cold artificial winter produces a color or pattern resembling that of a northern geographic variety of the same species, suggesting that the geographic "races" may not be genetically different, but are merely responding to different climates in their color development.

Effect of Light.—The best-known developmental influence of light is that upon wings in plant lice or aphids. These insects, in the parthenogenetic part of their reproductive cycle, may be either winged or wingless. Just how genes are related to wings is not known, but wings have a genetic basis, for different strains have different propensities to wing development, and they respond differently to light. One kind of aphid, under a given set of environmental conditions, may have wings in 50 per cent of individuals, another kind in only 2 per cent. In one strain of a certain species, aphids raised in continuous light are almost all wingless, while those reared in alternate light and darkness are almost all winged. The winged and wingless members of this strain are presumably genetically alike but are made phenotypically different by light conditions. Another strain of the same species reverses this response to light, in that more individuals are winged in continuous light than in alternate light and darkness, but it has been impossible to make all of them either winged or wingless by any external conditions. These two strains must differ in their genes, to account for this difference in their response to light.

Light produces a well-known darkening of the skin in human beings. Races of men are unlike in their skin pigmentation, and this distinction rests on differences in genes. While no amount of tanning of the skin of a white person could deceive any one into thinking that the darkened individual belonged to one of the colored races, the lack of confusion is partly due to the fact that races are distinguished by other things than skin color—shape of hair, features, etc. With respect to skin color alone, the influence of light on a white skin does tend to erase the racial distinction.

Nutrition.—In the common weed called the teasel, the stems are normally squarish. One variety, however, differs from the normal type by one mutant gene and has as a consequence a twisted stem (Fig. 84). Not all possessors of the twisted gene

have twisted stems, however. Plants must be rather well fed for the torsion to develop. Good soil, enough water and room, freedom from disturbance during growth, are means of providing this nutrition. Set the plants in sandy or gravelly soil, crowd them, withhold water from them, or transplant them two or three times, and the stems remain square even in the twisted variety. No such influence of nutrition can be exerted on the normal type. Despite any favorable treatment with respect to food, the common variety remains square-stemmed.

FIG. 84.- Portion of teasel plant showing twisted stem. To obtain many such plants, even in the variety characterized by twisted stems, it is necessary to nourish the plants abundantly. (*After DeVries, The Mutation Theory, Open Court Pub. Co.*)

In certain other plants, the number of repeated parts, such as the number of locules or chambers in the fruit, is increased by high nutrition. This example is not of the same kind as that of the teasels, however, for there is no variety of these plants which has the higher number of locules regardless of nutrition. The number of parts must be genetically determined, but there exist no other individuals of the same species having other numbers purely because of genes.

Many modifications of animals by differences in nutrition have been observed. A moth whose caterpillars normally eat only oak leaves may, by being forced to feed on walnut in these young stages, be made much paler. Bullfinches develop darker feathers if fed hemp seed. Green parrots of South America, if fed on the fat of catfish, become variegated with red and yellow. In none of these examples, however, does the environmentally modified individual resemble a different genetic variety; yet the characters changed are undoubtedly determined by genes.

Nutrition in developing embryos is capable of determining to some extent such general qualities as size, vigor, and general health. The advantage given to favored individuals in the developmental stages sometimes holds over to the adult. In mammals, this environmental modification may *appear* to change

the rules of heredity because of the source of the nutrition. The embryo is developed within the body of the mother, and the already digested food is received from her through the placenta and umbilical cord (Fig. 85). The mother may therefore influence the qualities of her offspring more than the father does in general physiological respects. Genetically the influence of the two parents is of the same degree except in sex-linked characters. But within the lifetime of the individual, particularly in early life, the mother may seem to be the more important.

FIG. 85.—Mammalian embryo in the uterus of the mother, showing mode of nutrition. *p*, placenta; *u*, umbilical cord.

Moisture.—A single example of the influence of moisture may be given. In Drosophila, each segment of the abdomen is marked by a dark stripe, extending across the body. In one of the mutant varieties of this fly, differing from the wild type in just one pair of genes, these bands of color may be interrupted, narrowed in places, or even lost. This mutation is called "abnormal abdomen." To possess these irregular markings, however, the fly must grow up in ordinarily moist conditions. If its surroundings are exceptionally dry, as in a very old culture bottle, the disturbances may largely or even wholly disappear, and the abdominal markings appear normal.

Chemical Substances.—Since development is largely a chemical phenomenon, it would be expected that unusual substances present during growth would modify adult form very considerably. This is true; but only rarely have any tests of this sort resulted in the production of an individual resembling a different genetic variety, that is, one due to a known difference in genes regardless of the chemical environment. Perhaps this paucity of examples is due to the fundamental importance of chemistry in biological matters. It is hard not to make the chemical change a radical one. Thus, when sea urchin or snail eggs are raised in chemical solutions only very slightly different from sea water, their embryos may develop in a variety of abnormal ways; but the changes are eventually fatal. In fishes, exposing the eggs to very weak solutions of certain sub-

stances causes them to develop one-eyed monsters. Other animals have been made double-headed by comparable treatment. If chemical treatment could be made less extreme and could be effected with substances more closely related to those in protoplasm, it is probable that one genetic variety could be transformed into the phenotype of another genetic type with considerable frequency.

An approach to this imagined success has been made in certain aquatic crustacea by putting small quantities of salts into the water in which they live. These animals have different forms of body, including a beak or protrusion of the head, in different strains. To some extent these differences are genetic, since the strains differ with respect to them under like conditions, though in no instance is it known how many pairs of genes are concerned in the distinction. Changing the salt content of the water has changed members of a long-beaked strain, for example, so as to make them resemble those having a short rounded beak.

Suggestive of possible change of inherited characters are some experiments with α-dinitrophenol on development of the frog (Dawson 1938). Very weak solutions of this substance retard development of the gills, eyes, and intestines. No inherited normal variation of the frog has been imitated by this treatment, but since rates of development have an influence on the end result (page 53), such imitation would seem to be a possibility.

Hormones.—Probably the best success in chemical interference with genetic characters has been attained by the use of hormones in the vertebrate animals. Hormones are substances produced in ductless or endocrine glands (Fig. 86), or perhaps by tissues in general. From these glands or other places of origin, whatever their nature, they diffuse into the blood. There they are carried about, come into contact with many different organs or tissues, and, to some of these organs, serve as chemical messengers. The hormones control in some degree the activities of those parts which are responsive to them. In adult animals, physiological processes are thus controlled; in embryos, development is in part directed by them.

Important endocrine glands are the thyroid, the parathyroids, the thymus, the adrenal bodies closely associated with the kidneys, pineal body, spleen, and the pituitary at the base of

the brain. Certain other organs best known for other functions also produce endocrine secretions. Thus the principal reproductive organs, the ovaries and testes, secrete substances directly into the blood, as also does the pancreas whose best understood function is the production of a digestive fluid.

The functions of these glands are exceedingly variable. The thyroid shares in control of the rate of basal metabolism, accelerates growth, and regulates differentiation. Through these capacities, it normally prevents such defects as cretinism and myxedema. Metamorphosis in frogs and salamanders, or the

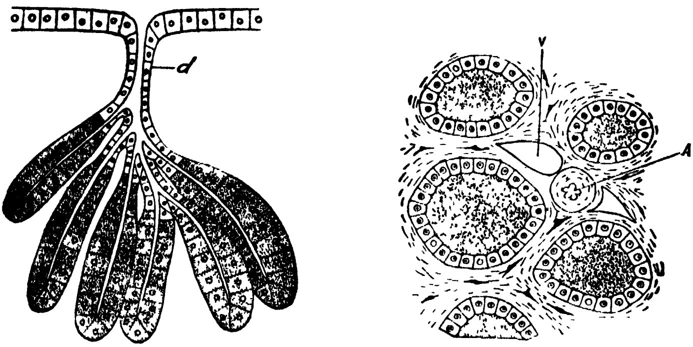


FIG. 86.—Two kinds of glands. At the left, gland with duct (d) through which the secretion is discharged into some cavity or upon a surface. At the right, part of a ductless gland, the thyroid, whose secretion leaves the gland by diffusion into the blood carried by the arteries (A) and veins (V) scattered through the organ. Glands of the latter type are known as endocrine glands.

transformation of the tadpole into the adult, is dependent on a proper thyroid secretion. Iodine has an important, though vaguely defined, influence on the activity of the thyroid and is used to alleviate certain types of goiter. The parathyroid glands, closely associated with the thyroids, regulate calcium metabolism, including the quantity of calcium in the blood. The pituitary secretion causes a prolonged rise of blood pressure, because of contraction of the arterial walls, and is used clinically to prevent surgical shock. It stimulates growth of hair and increases growth especially of the hands and feet. It also controls the elimination of water by the kidneys and is of service in treating the disease diabetes insipidus, in which excessive amounts of urine are produced. Adrenalin causes a rise of blood pressure and is used in medical practice to prevent superficial hemorrhage. Its effect on involuntary muscles elsewhere is various, for it

dilates the pupil of the eye, excites the muscles of the uterus in some animals but inhibits their contraction in others, and stops muscular movement or reduces the tone (state of chronic contraction) of the intestine. The adrenals and pituitary together govern color changes in frogs and other amphibia, by expanding or contracting the pigment in certain cells of the skin. Insulin, which is produced by certain groups of cells in the pancreas, governs sugar metabolism and is important in the prevention of diabetes mellitus. The thymus is associated with the function of reproduction, since it degenerates at sexual maturity and persists in castrated animals. Extract of the thymus reduces blood pressure and accelerates heart beat.

The ovaries and testes produce internal secretions which influence the development of secondary sexual characters, to be described in a later chapter.

The various endocrine glands are known in a few cases to influence one another in important ways. Thus, the pituitary stimulates the growth of the thyroid, an interplay of several hormones directs the reproductive cycle of mammals, and it is likely that many other such interrelations exist.

Hormone Modification of Inherited Characters.—Among the things altered by endocrine secretions are some that have a known and sometimes fairly simple genetic basis. Cretinism, some kinds of goiter, hair growth, blood pressure, diabetes insipidus, and diabetes mellitus are in some degree inherited, as described in a later chapter, and their dependence on certain hormone conditions indicates the important relation which these substances have to heredity.

Endocrine racial types have been distinguished by certain anthropologists, who describe the Caucasian as marked by strong development of the pituitary and adrenals, the Negro as deficient in adrenals, and the Mongolian as defective in the thyroid. Certain biologists have suggested that such features as slenderness or stockiness of build, baritone or tenor voice, alert or sluggish mentality, and long or round head are dependent on whether the thyroid is highly effective or deficient. Along with this suggestion goes the companion notion that if the food of an individual habitually includes much iodine-containing material, the activity of the thyroid is thereby modified, and the corresponding structural changes follow. Sea

foods in general contain more iodine than do the foods common in most inland regions, and those who see in the thyroid an important agency in the production of certain physical and mental characteristics are not at a loss for examples of corresponding differences between coast dwellers and those far from the ocean.

Since racial characters are inherited characters, the supposed dependence of race distinctions upon hormones would constitute chemical modification of genetic characters.

Internal Environment.—The question may be raised whether, with respect to endocrine secretions, we are or are not dealing with the environment. Are they not merely part of the physiological mechanism through which the pertinent characters are developed? If the hormones themselves are governed by genes, any characters which they modify are as purely genetic as are other characters in whose development the intermediaries are not known. The hormones would constitute an internal environment, but one which, if the above supposition were correct, would be wholly controlled by the genotype.

The endocrine secretions have, however, some variability which is, or may be, independent of the genes. They are not always present in the same quantity, nor probably have they always the same quality. Some of them are notably influenced by the diet and by mental and other physiological states. The mode of life, degrees of comfort or hardship, would therefore have an important relation to hormones and hence to development. Furthermore, the essential chemical principles of these secretions may be extracted and administered in medical practice. Important means of artificial control of inherited characters are thus afforded.

Heredity versus Environment.—The knowledge that genetic characters are modified by environmental agencies, even to the extent of making them resemble other genetic characters, should effectively dispose of any notion that a quality (phenotype) must be either hereditary or environmental. One still hears asked the question whether this or that trait is hereditary or environmental, with the implication that these categories are mutually exclusive. The fact is, it may be both. A quality may properly be said to be inherited if it has a genetic basis of any sort, no matter how much it may be influenced by external agencies. If two individuals, in their development, respond

differently to the same environment, it is usually safe to say that the respect in which they differ is a genetic quality. Even if environment is much more influential than the genes, the character is still hereditary. And no matter how little environment changes development, any trait thus modified is environmental—as well as genetic.

Qualities differ enormously in their degree of dependence on external factors. Eye color in man, as in the other mammals, is almost wholly fixed by the genes. Fingerprint patterns, short of actual mutilation of the skin, are probably nearly free of any environmental influence, except that which resides within the individual. Stature, on the contrary, can be considerably modified by treatment sufficiently early. General vigor is likewise so governed in large part, and conditions bad enough may easily undermine even the most perfect physique.

It is seldom profitable to estimate the relative dependence of a character on heredity and environment. To say that a quality is 70 per cent inherited has only a statistical significance, and might be of use to an insurance company when writing policies. More is to be gained by discovering the *way* in which both heredity and environment influence it. This information, if gained, can then be applied to individual cases.

CHAPTER XVII

CHANCE AND HEREDITY

So many of the events connected directly or indirectly with heredity occur in a purely or approximately random way that it is necessary to comprehend what is involved in chance. The word chance is a much misunderstood term. An event that is due to chance is not, as is so often apparently believed, without a cause or group of causes. Nothing ever really happens without cause. When however, the causes are unknown, or beyond the possibility of control, and when they operate in such a way as to produce a variety of results without bias in favor of any one result, the effect is said to be due to chance. It is not a matter of accident, for example, whether a coin, at a given throw, turns head or tail; something in the motion of the hand of the thrower determines which way the coin shall fall. But the thrower does not know what feature of his movement determines the result of the toss and could not control it if he did know. As a result of such unknown and uncontrolled causes, the coin falls in the long run with one side up as often as with the other, provided it has perpendicular edges and sharp corners and is thrown on a smooth hard surface. If dice have six equal faces and all of their edges are equally rounded, and if they are thrown in the usual manner, one face turns up as often as any of the others (Fig. 87). This result is said to be due to chance, though in any individual toss the combined impulses given any die inevitably turn up a particular face. If all the faces are equal and all the edges are of the same shape and the die falls distinctly more often upon one face than upon the others, that is evidence that the die is loaded;

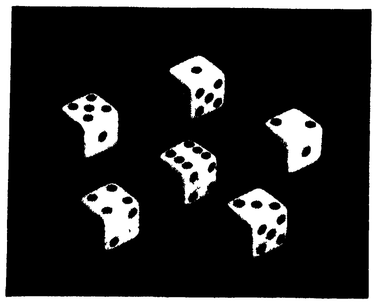


FIG. 87.—The six possible positions of dice. If the cubes are perfect, each position should occur as frequently as any of the others in a large number of throws.

that is, the center of gravity is not in the geometrical center of the cube. In these latter examples chance is not alone in determining the result of the toss.

Nothing here said is intended to imply that in a given *finite* number of trials the various possible results will occur with equal frequency. If the number of trials is small, some of the possible results may occur much more often, or much less often, than the theory of chance would lead one to expect. The theory states the results expected in an infinite number of trials. In any smaller number of trials, there is likely to be some deviation from the expectation; and the smaller the number of trials the greater the probable relative deviation.

Chance, in the sense explained above, plays an important part in the behavior of the chromosomes and consequently in the distribution of the genes to the germ cells and in the union of the male and female germ cells in fertilization. The ratios obtained in breeding experiments are largely dependent on these fortuitous events. From these ratios are derived the answers to questions on which hinge matters of human welfare and happiness. Particulars of some of these relations are presented in the remainder of this chapter.

Chance and the Pairs of Chromosomes.—When the homologous chromosomes have paired in the immature germ cells and the spindle of the reduction division is formed, the chromosome pairs take their position upon the equator of the spindle partly by chance, that is, while the maternal chromosome is turned toward one end of the spindle and the paternal chromosome toward the other end, it is a matter of chance *which* end of the spindle is faced by either of them. This means that, although a given pair of chromosomes must be placed a certain way as a result of the forces that move it about, if thousands of pairs are acted upon by similar forces, half of them may be expected to turn one way, and half of them the other way.

As an illustration, assume that there are five pairs of chromosomes in a cell and that for convenience these pairs be numbered one to five. Though all of these pairs take their place on the spindle of the dividing cell at the same time, for the purpose of this discussion it may be assumed that they do so in succession. When the first pair moves to the equator of the spindle, the maternal member may turn either to the right or to the left.

The chance that this maternal chromosome will turn to the right is 1 in 2 or in other words is ½. After it has taken this position with the maternal chromosome (white) to the right (as in Fig. 88, *a*), assume the second pair of chromosomes to take its place on the equator of the spindle. Again the chance that the maternal chromosome will turn to the right is 1 in 2 or one-half. The other half chance is that the maternal chromosome will turn to the left; and the total of all chances is 1. Considering both pairs of chromosomes simultaneously, therefore, the chance that *both* of them will have their maternal members to the right (as in Fig. 88, *b*) is ½ × ½, that is, ¼ or 1 chance in 4. The three other of these four chances are represented in *c*, *d*, and

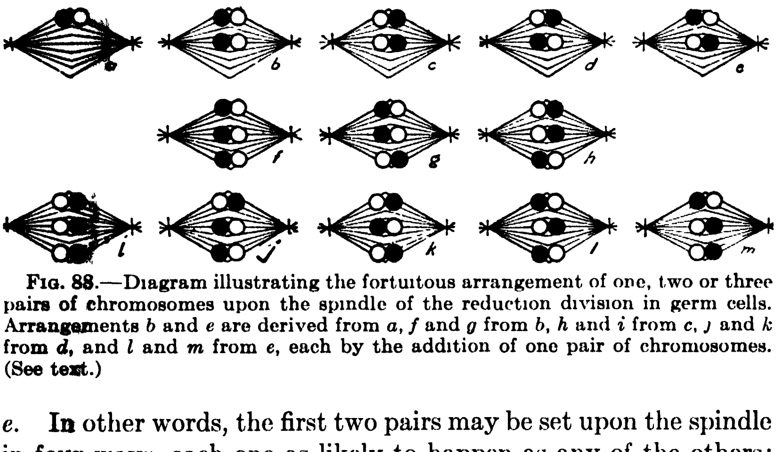


FIG. 88.—Diagram illustrating the fortuitous arrangement of one, two or three pairs of chromosomes upon the spindle of the reduction division in germ cells. Arrangements *b* and *e* are derived from *a*, *f* and *g* from *b*, *h* and *i* from *c*, *j* and *k* from *d*, and *l* and *m* from *e*, each by the addition of one pair of chromosomes. (See text.)

e. In other words, the first two pairs may be set upon the spindle in four ways, each one as likely to happen as any of the others; hence the chance or probability of the occurrence of *each* of these combinations is ¼. Of 100 spermatocytes undergoing reduction division, about 25 should show the combination of any two of their pairs of chromosomes illustrated in *b*, 25 the combination shown in *c*, 25 that shown in *d*, and 25 that in *e*.

Let a third pair of chromosomes now take its place on the spindle, and the chance that its maternal chromosome will be placed on a given side is again ½. If the first two pairs be placed as in *b* (whose chance of occurrence is ¼), the combination shown in *f* by the addition of a third pair should occur in ½ × ¼ of all cases or ⅛. The third pair might have been turned the other way, as in *g*, and this combination has a chance or probability likewise of ⅛. The third pair might have been added

to the combinations shown in *c*, *d*, and *e*, resulting in six more combinations illustrated in *h* to *m*, inclusive. Each of these combinations is as likely to occur as any of the others, that is, three pairs of chromosomes may be arranged in eight $(2 \times 2 \times 2)$ ways, and the chance that any one of them will result in a given cell is $\frac{1}{8}$. In a thousand spermatocytes, any three pairs of chromosomes should be arranged with all maternal chromosomes to the right (*f*), for example, in about 125 cases.

In the preceding discussion, the results stated are those expected from consideration of the theory of chance. Fortunately, the correctness of the conclusions is demonstrated by observations by Carothers and others upon the chromosomes of

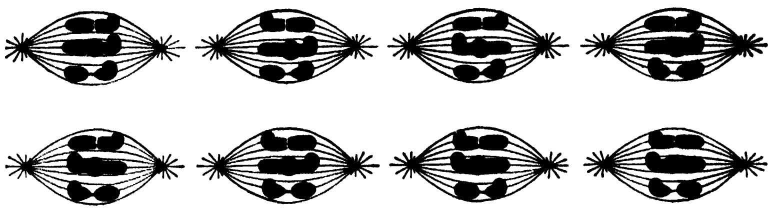


FIG. 89.—Fortuitous arrangement of chromosome pairs on the reduction spindle of spermatocytes of grasshoppers. In each of three pairs, one chromosome was bent in all spermatocytes of the same animal. It is possible to arrange the three pairs in eight different ways with reference to one another, and all these arrangements were observed in actual cells. Moreover, the cells exhibiting the eight different arrangements were approximately equally numerous. The bent chromosome could be either maternal or paternal in different matings. (*After Miss Carothers.*)

grasshoppers. In certain individual animals the chromosomes constituting several pairs were not alike in shape. One chromosome of each pair was bent, apparently owing to the pull of the spindle fibers upon them. All spermatocytes in the same individual had the same peculiarity.

In a grasshopper in whose cells there are three pairs of unlike chromosomes the reduction spindle should show eight different arrangements, as in Fig. 89. All of these were found in actual cells and in about the same number of cells for each combination. This is an excellent confirmation of the expectation derived from theoretical considerations. It indicates that the placement of the chromosomes is random.

It should now be clear that where five pairs of chromosomes are involved there are $2 \times 2 \times 2 \times 2 \times 2$, or 32, possible combinations, of which each has 1 chance in 32 of happening. That

combination which has all five maternal chromosomes to the right should therefore occur in about 100 cases out of a total of 3200 spermatocytes.

Since most animals have more than five pairs of chromosomes, the possible combinations of pairs on the reduction spindle are much more numerous than 32, and the chance that any one of them happens is correspondingly smaller. Each time a pair of chromosomes is added, the number of possible arrangements in the reduction division is doubled, and the chance that any one arrangement will occur is halved.

Although the arrangement of the chromosome pairs on the reduction spindle is almost always random, as described in the foregoing account, there are a few instances in which the position is definitely fixed in relation to some other event. In the fly Sciara, for example, the pair of chromosomes, in which the gene for fused wing veins and its normal allele are located, always takes the same position in relation to the size of the cells produced. The reduction division in the males is peculiar in that one large and one small cell are produced, and only the large cell survives. The pair of chromosomes in question is so turned that its maternal member goes into the large cell; hence only the maternal character is transmitted. In teosinte, a plant related to corn, the assortment of the chromosomes in reduction is not wholly random. In a certain moth it has been claimed that the position of one pair of chromosomes in the production of the eggs could be influenced by temperature. Since these chromosomes were the ones associated with sex, the sex ratio was thereby altered. These instances of wholly or partially determinate distribution of the chromosomes are uncommon, however, and the bulk of the assortment of chromosomes is fortuitous.

Combinations of Chromosomes in the Germ Cells.—If the principle of chance as described in the preceding section is thoroughly understood, some of its consequences will be comprehended without difficulty. The student of heredity is particularly interested in the combinations of maternal and paternal chromosomes that may pass together to the same germ cells, for upon these combinations depend the combinations of grandparental genes in given individuals. That arrangement of chromosome pairs in which all the maternal members are on the right at the reduction division was shown above to be expected once in 32 times when

five pairs of chromosomes are involved. By the same sort of argument, it may be shown that any other definite combination also occurs 1 time in 32. Thus, the first pair will have its maternal member to the right and the four other pairs their maternal chromosomes to the left 1 time in 32. Likewise, the second and fifth pairs may have their maternal chromosomes to the right while the first, third, and fourth are placed with the maternal members to the left 1 time in 32. Each other possible arrangement of chromosome pairs theoretically has the same frequency, namely, 1 time in 32.

The application of these operations of chance in heredity are obvious, since the genes are in the chromosomes. If only one pair of characters is being studied, the calculation may concern itself only with the one pair of chromosomes in which the genes for these characters reside. In this case there are only two possible positions of the chromosome pair on the spindle of the reduction division, and only two kinds of germ cells can result from that division. If two pairs of characters whose genes reside in different pairs of chromosomes are being studied, only these two chromosome pairs need be considered. It is clear that there are 2×2 possible arrangements of the two chromosome pairs on the spindle. In an individual that is heterozygous for both pairs of characters (as an F_1 animal), this means that 2×2, or 4, kinds of germ cells will be produced. If three pairs of characters are being simultaneously studied and their genes are in three different pairs of chromosomes, only these chromosomes need be considered. These three pairs of chromosomes, obviously, can be arranged in $2 \times 2 \times 2$ different ways on the spindle of the reduction division, which means that an individual which is heterozygous for all three of the pairs of characters produces germ cells of $2 \times 2 \times 2$, or 8, different kinds with respect to these characters.

It must be clear from the above that if five pairs of characters (whose genes are in five pairs of chromosomes) are considered simultaneously, the number of chromosome arrangements (and hence the number of kinds of germ cells produced by a fully heterozygous animal) will be $2 \times 2 \times 2 \times 2 \times 2$, or 32. The student can now readily calculate how many chromosome combinations and hence how many resulting combinations of maternal and paternal hereditary factors are possible as soon as the number

of pairs of chromosomes (or of independent genes) is stated. It is always 2 raised to the power whose exponent is the number of independent characters for which the animal is heterozygous. These computations of numbers of kinds of germ cells have been made once before but are repeated here to emphasize their dependence on chance.

It must not be forgotten, furthermore, that all the combinations possible in a given example are equally abundant. If, as in the case of five pairs of chromosomes or genes, 32 combinations occur, each of them should appear in the long run once in 32 cells. An animal heterozygous for two pairs of characters (with genes in different pairs of chromosomes) therefore produces four kinds of germ cells in equal numbers. An animal heterozygous for three pairs of characters (with genes in different pairs of chromosomes) produces eight kinds of germ cells, each kind as numerous as any of the others.

This assumption of numerical equality of the several classes of germ cells does not rest merely on the theory of chance; it has been demonstrated by testcrosses to be approximately correct. Figure 75, relating to color of eyes and ocelli in Drosophila, furnishes such proof for an animal heterozygous for two pairs of genes (hence producing four kinds of germ cells), and comparable experiments are on record for organisms heterozygous for larger numbers of genes.

Chance and the Union of the Germ Cells.—When the eggs are of several kinds, or the spermatozoa are of several kinds, or when both eggs and spermatozoa are of different kinds, new opportunities are offered for the operation of chance in the union of egg and spermatozoon in fertilization. In the experiment illustrated in Fig. 75, four kinds of germ cells were produced, and it was tacitly assumed that all the F_1 germ cells entered into testcross offspring. If the F_1 fly was a female, it is not improbable that all of its eggs actually were fertilized. Under this circumstance no element of chance enters into union of egg and sperm, for the four classes of testcross flies follow directly from the four kinds of F_1 eggs. Flies and eggs are in fact, except for the added spermatozoa, the same individuals. Many animals, however, deposit their eggs in such a way that only part of them are fertilized. In such species, if the spermatozoa are all of one kind, the only way in which chance operates is in determining

which eggs remain unfertilized. If there are four kinds of eggs, equally numerous, it would be expected that the eliminated eggs would come equally from all of the four classes. Any other result than this would mean that something else than chance, or something in addition to chance, was determining which eggs were fertilized. Such aberrant results have been discovered in a few cases, and are attributed to "selective fertilization," meaning that certain kinds of eggs are fertilized more often than other kinds. The reason for the discrimination is usually not known.

When the spermatozoa are of several kinds and the eggs all alike, there is always opportunity for the operation of chance, for the spermatozoa are always so numerous that most of them can never fertilize eggs. Indeed, in some animals millions of spermatozoa are produced for every one that fertilizes an egg. Unless selective fertilization affects the fate of the spermatozoa, those spermatozoa that happen to fertilize eggs must, as a matter of chance, come from the several classes of spermatozoa in proportion to their number.

When both spermatozoa and eggs are of several kinds, a greater variety of combinations due to chance is possible. A simple situation of this kind is found in the mating of two F_1 animals in which only one pair of characters is concerned. Each animal is heterozygous, and hence produces two kinds of eggs or spermatozoa. Eggs and spermatozoa can, therefore, meet in four possible ways (AA, Aa, aA, aa), all equally abundant. Two of these ways (Aa and aA) are identical, so there are only three combinations, of which one (Aa) is twice as numerous as the others (AA and aa). When two individuals heterozygous for two pairs of characters are mated, four equally numerous kinds of eggs are fertilized at random by four equally numerous kinds of male cells. The 16 combinations are consequently equally abundant. These combinations are not, however, all different. An inspection of the table in Chap. XIII showing the genotypes of the F_2 generation from a cross involving color of fruit and shape of fruit in squashes will show, as was there pointed out, that there are only nine different genotypes. There are four of one kind, two of each of four other kinds, and one of each of the remaining four genotypes.

This discussion need go no further than to point out that when three pairs of characters are involved, the fortuitous unions

of eight kinds of eggs and eight kinds of spermatozoa afford a still greater variety of combinations, 64 to be specific. An F_2 generation of this sort includes 27 genotypes which, if there is dominance in each of the pairs of genes, fall into eight phenotypes. Such generations have already been tabulated in very condensed forms showing principally the phenotypes in Chaps. XIII and XV.

It should be made clear that in the discussion in this section the fortuitous events referred to have nothing to do with the formation of the germ cells and the combinations of genes collected in them. That matter was the subject of the preceding section. The chance referred to in the present section is that which has to do with the union of different kinds of eggs and spermatozoa. The diversity due to chance in the *formation* of germ cells is thus multiplied by the diversity due to chance in the *union* of germ cells in fertilization.

Chance and the Mendelian Ratios.—It must have occurred to the student during the discussion above that the characteristic ratios obtained from various matings are the results of chance. The 1:2:1 ratio characteristic of the F_2 generation when dominance is lacking is due to the fortuitous union of two kinds of eggs and two kinds of spermatozoa. The 3:1 ratio is but a modification of 1:2:1 and is likewise the result of chance. The 1:1 ratio of the backcross is the result of the random fusion of two kinds of eggs (for example) with one kind of spermatozoon. The 9:3:3:1 ratio results when four equally numerous kinds of eggs are fertilized by chance by four equally numerous kinds of spermatozoa, as in the production of the F_2 generation from a cross involving two independent pairs of characters. The 1:1:1:1 ratio illustrated in Fig. 75 is due to the random union of four kinds of eggs (for example) with one kind of spermatozoon.

Being the results of chance, these ratios are seldom precisely realized in actual experiments; but the deviations from the expectation are as often in one direction as in the other. How great these deviations may be without indicating that anything else than chance is determining them may be computed mathematically but a discussion of these deviations belongs to an account of statistical methods which is briefly undertaken in the Appendix. Such computations show that a ratio of 45:55, for example, may easily represent a genuinely 1:1 ratio.

Chance Eliminated in Certain Processes.—Lest the student be misled into thinking that most processes connected with heredity are the results of fortuitous events, it may be pointed out that some things can happen in only one way. The pairing of the chromosomes early in maturation is one such process. Each chromosome can pair with only one particular chromosome, namely, the other chromosome having genes for the same sorts of characters. Also, there is no chance in the *equation* division. The chromosomes are *duplicated* at this division, not merely separated as in the reduction division. The new chromosomes thus produced are presumably equal, and the two daughter cells are therefore alike in all respects.

Certainty in Spermatozoa, Chance in Eggs.—In one particular the eggs and spermatozoa differ markedly in their liability to chance. This is in the numbers of the different kinds of germ cells produced by an animal which is heterozygous for only one pair of genes. In the male, theoretically exactly the same numbers of spermatozoa of the two kinds are produced. At every reduction division one of the chromosomes containing the genes for this particular pair of characters goes to one germ cell, while its homologous chromosome goes to the other cell resulting from this division. The numbers of the two kinds of cells produced must, therefore, be absolutely equal. If a million spermatocytes undergo reduction division, there result necessarily precisely a million cells of each of the two kinds. The above is true only when but *one* pair of characters is involved. When two or more independent pairs of characters are concerned, the numbers of the four or more kinds of spermatozoa are due to the fortuitous position of the chromosome pairs *in relation to each other.*

Unlike the spermatozoa, however, the eggs of an animal heterozygous for a single pair of characters are not necessarily of equal numbers in the two classes. The reason for this difference is that in maturation of eggs only one cell of the four potentially arising from the two divisions ever becomes functional. The other three (more often two) are polar bodies. Chance, therefore, has an opportunity to cause differences in the number of the two kinds of eggs produced. If, by accident in the oöcytes in the pair of homologous chromosomes containing the factors for which the animal is heterozygous, the maternal chromosome

should turn so as to enter the large functional daughter cells more often than the minute polar bodies, more eggs would be produced containing this chromosome than eggs containing the homologous paternal chromosome. If the polar bodies were to be counted as well as the functional cells, in making the above comparison, the result would be precisely 50 per cent of each kind, just as in the male.

In practice the distinction just made between eggs and spermatozoa does not exist; for, while all cells in the male maturation have the normal possibility of functioning (four spermatozoa from each primary spermatocyte), never do all such cells survive. In every male animal some of the prospective germ cells die or fail to complete normal development. Consequently, even in an individual heterozygous for just one pair of genes, the numbers of the two kinds of spermatozoa need not be, and probably never are, exactly equal. It is worth noting, however, that the reason for slightly different numbers when equal numbers are expected is different for the two sexes. In males the inequality depends on accidental or other failure to develop; in females it is traceable to polar bodies and chromosome positions, plus occasional failure to develop.

Practical Applications of Knowledge of Chance.—Aside from the understanding it affords of the processes connected with heredity, knowledge of the chance involved in any particular situation may be of value to the practical breeder in determining his breeding procedure or to human beings in guidance of their conduct. When a breeder desires to create a certain type of animal or plant by recombination of characters previously existing only in separate strains, he must usually select certain individuals out of an F_2 or later hybrid generation. How ambitious his breeding plans must be depends on how rigidly he must select. If he is selecting for a single character, and the kind of organism he wishes may be expected to make up one-fourth of the progeny, a generation including one or two dozen individuals is very likely to provide him the required type. If, however, he seeks to combine a number of characters and only 1 in 64 or 1 in 256 will have the right combination, the number of individuals must reach hundreds or even thousands to insure success. In such instances it may be wiser to accept a partial approach to the desired combination and to make further crosses

with it. The whole process could thus entail less labor, though more time.

In human affairs, the probability that a certain individual carries a given recessive gene is often important in deciding the advisability of marriage. Some practice in determining this probability from family histories is desirable. Suppose that the person in question has some feeble-minded relatives, but that he himself is apparently wholly normal. Assume that feeble-mindedness is a simple recessive, which is not quite true. The known family history is that shown in Fig. 90, and the subject of inquiry is the man numbered 3. It is desired to know what chance he has of carrying the feeble-minded gene (f). Now, the fact that No. 2 is feeble-minded shows, under the assumption that this defect is a simple recessive, that both parents are heterozygotes. Consequently, their phenotypically normal children will be of two genotypes; $\frac{1}{3}$ of them ($\frac{1}{4}$ of the whole family) should be homozygous normals (FF) while $\frac{2}{3}$ of them (half of the whole family) should be heterozygous normals (Ff). Number 3, like each of his brothers, has therefore two chances in three of carrying the feeble-minded gene.

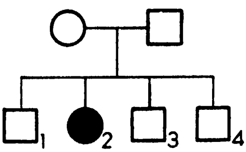


FIG. 90.—Family history illustrating chance. The blackened individual is feeble-minded. What is the probability that her brothers and sisters are heterozygous for this defect?

Sometimes the policy to be decided is whether cousins should marry. The general family to which they belong has produced an occasional individual possessing a recessive trait which it is not desirable to pass on. What is the chance that the cousins would have a child burdened with it? A concrete problem is presented in Fig. 91, in which the unfortunate recessive persons are shown in black. Individuals III-3 and III-6 contemplate marriage. Though they are normal, may not a child of theirs exhibit the character they fear?

In answering this question, it is first to be noted that, in order that any of their children might be homozygous recessives, both III-3 and III-6 must be heterozygotes. One must decide first, therefore, what their chances of being heterozygous are. Consider III-3 first. One of her brothers is recessive, hence their parents (II-1 and II-2) must both be heterozygous. Any normal child of II-1 and II-2 has in consequence two chances in three

(a $\frac{2}{3}$ chance) of being heterozygous, as was just explained for feeble-mindedness in a similar situation. The probability that III-3 is heterozygous is thus $\frac{2}{3}$. Turn now to III-6. It is known that his mother (II-3) is heterozygous for she had a recessive mother; no chance is involved in this feature of the family history. Her husband (II-4) we will assume is a homozygous normal, because his family history has been examined and no relatives found to possess the character in question. With these parents, III-6 has therefore a $\frac{1}{2}$ chance of being heterozygous.

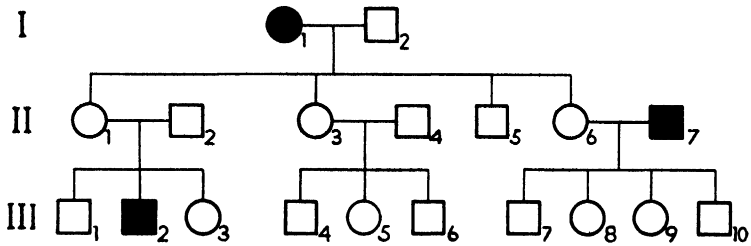


FIG. 91.—Family history illustrating chance. The blackened individuals exhibit an undesirable recessive character. What risk of transmitting this character would inhere in cousin marriages within the last generation?

Since III-3 has a $\frac{2}{3}$ chance of being heterozygous and III-6 has a $\frac{1}{2}$ chance of being heterozygous, the chance that *both* of them are heterozygous is $\frac{2}{3} \times \frac{1}{2}$, or $\frac{1}{3}$. There is thus one chance in three that both of the cousins contemplating marriage are heterozygous. Now, when both parents are heterozygous, any child of theirs has a $\frac{1}{4}$ chance of exhibiting the recessive character. Inasmuch as the chance that both parents are heterozygous is $\frac{1}{3}$, the chance that any given child of theirs will be recessive is $\frac{1}{3} \times \frac{1}{4}$, or $\frac{1}{12}$. Each child they produce has 1 chance in 12 of showing the undesirable quality. If they have a family of 4, there are 4 chances in 12 that one child will be recessive.

For drill, the student is encouraged to determine what chance a child of III-5 and III-7 would have of being recessive. To verify his solution, the answer may be given as 1 in 8.

CHAPTER XVIII

LINKAGE

In earlier chapters, when two pairs of genes were simultaneously studied, examples were so chosen that genes would be distributed to the germ cells independently. As a consequence of their random recombination in the reduction division, the several classes of germ cells produced by a heterozygous organism were equally numerous, and the phenotypic dihybrid ratio in F_2 was 9:3:3:1. When in some of these examples the genes interfered, or cooperated, with each other in the production of their characters, their distribution to the gametes was still free and random, and the F_2 ratio was some modification of the 9:3:3:1 ratio, such as 9:3:4 or 9:7. All this independence rested on the fact that the two (or more) pairs of genes were in different pairs of chromosomes, and the chromosomes are independent in their placement on the reduction spindle.

Linkage in Male Drosophila.—The story is very different when the two pairs of genes are in the same pair of chromosomes. What happens will be better understood from concrete examples, and the first is taken from among the numerous characters of Drosophila. One pair of genes concerns the shape of the wings; the wild-type fly has flat wings which rest horizontally, the mutant type curled wings (Fig. 92). The other pair governs the color of the ocelli, the wild type being reddish yellow, the mutant variety white (Fig. 75).

These two pairs of genes are in the same pair of chromosomes, namely, the so-called third pair. The genes for shape of wings are located about the middle of these chromosomes, those for color of ocelli about halfway between the middle and one end, as diagrammatically illustrated in Fig. 93. Suppose that the mutant genes are matched against their wild-type alleles by crossing a fly having flat wings and yellow ocelli (the wild-type characters) with one having curled wings and white ocelli, as in Fig. 94. In accordance with a common practice already outlined

in an earlier experiment, only the mutant genes are marked in the chromosomes. Any chromosome unmarked is thus shown to contain only wild-type genes, with respect to all loci under consideration. The eggs of the wild-type female (left) thus have only wild-type genes; the spermatozoa of the male (right) have the genes for curled and white ocelli in the same chromosome.

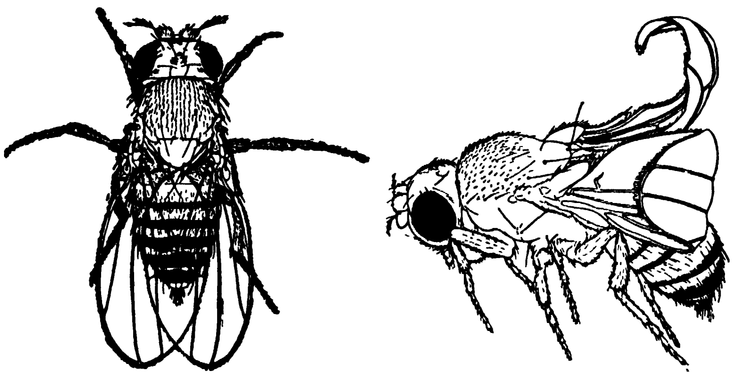


FIG. 92.—Wild-type Drosophila, left, and its curled-wing mutation, right. (*From Morgan, Bridges and Sturtevant in Bibliographia Genetica.*)

In the offspring, therefore, one chromosome of this pair has the two mutant genes *cu* and *wo*, while the other has no mutant genes (that is, it contains *Cu* and *Wo*). These F_1 flies have, of course, flat wings and yellow ocelli.

A male is now chosen from the F_1 generation, and mated with a doubly recessive female out of the curled white stock. She has the genes *cu* and *wo* in both of her chromosomes of the third pair. This homozygous female produces only one kind of egg, *cu wo*, with both of these genes in the same chromosome. The F_1 male shows now the effect of linkage. While in all examples so far presented an individual which is heterozygous for two pairs of genes produces four kinds of germ cells, this male produces only two kinds of spermatozoa. At the reduction division the chromosomes separate, the wild-type chromosome going to one spermatocyte, the *cu wo* chromosome going to the other. Two, and only two, kinds of spermatozoa are thus produced; one is

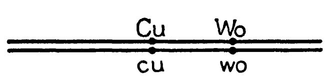


FIG. 93.—Chromosome pair 3 in Drosophila with the genes for curled wing (*cu*) and white ocelli (*wo*) and their wild-type alleles.

wild type (Cu Wo), the other cu wo in composition. They should be equally numerous.

When these two kinds of spermatozoa fertilize the one kind of egg, two kinds of testcross offspring are produced. One kind has the wild-type characters, flat and yellow, and is heterozygous; the other kind is curled and white. They are present in about the same ratio as were the two kinds of spermatozoa, namely, 1:1.

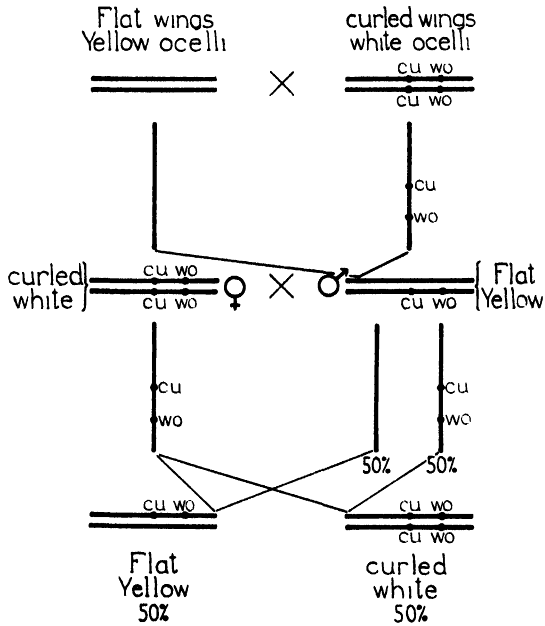


FIG. 94.—Linkage of genes for curled wing (*cu*) and white ocelli (*wo*) in Drosophila, as illustrated by a cross between a doubly heterozygous F_1 male and a doubly recessive (hence homozygous) female. Wild-type genes are not marked.

Linkage in Female Drosophila.—Some modification of the linkage phenomenon is shown by the female. To illustrate this difference, let the cross involving curled wing and white ocelli be repeated; and to introduce variety, use a female having white ocelli (otherwise wild type, hence with flat wings) and a curled male (otherwise wild type, that is, with yellow ocelli). The cross is illustrated in Fig. 95. The eggs of the female are all *wo*, the spermatozoa of the male all *cu*. The F_1 flies are therefore heterozygous for both genes, hence are wild type, with flat wings

and yellow ocelli; but *wo* is in one chromosome 3, *cu* is in the other chromosome of the same pair.

To proceed with the linkage test, let a female F_1 be mated with a doubly recessive male (*cucu wowo*). The male produces only one kind of spermatozoon (*cu wo*). The doubly heterozygous female, however, produces several kinds of eggs. Her chromosomes of pair 3 separate without change in the reduction

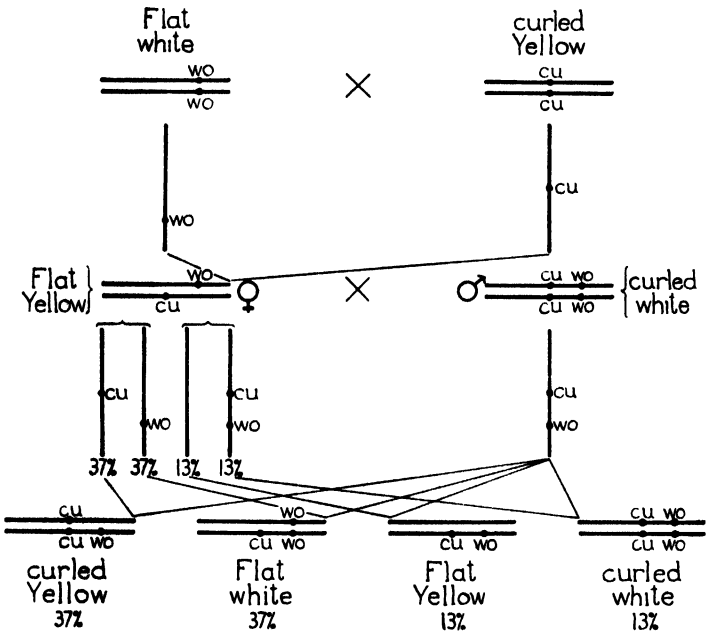


FIG. 95.—Linkage of genes for curled wing (*cu*) and white ocelli (*wo*) in Drosophila, as illustrated by a cross between a doubly heterozygous F_1 female and a doubly recessive (hence homozygous) male. The chromosomes remain intact in 74 per cent of the oöcytes, break and recombine in the other 26 per cent.

division in most of the oöcytes, and there result some eggs containing *cu* and others with *wo*, in about equal numbers. Not all the oöcytes behave this way, however. In some of them the chromosomes of pair 3 effect an exchange, such as is diagrammatically shown in Fig. 96. The two chromosomes break at some point between the locus of *cu* and that of *wo*, and the pieces are recombined. As newly constituted, the one chromosome contains both *cu* and *wo*, while the other has both wild-type alleles. When cells that have experienced this exchange undergo

reduction, two more kinds of eggs are produced, namely, wild type and *cu wo*.

About 74 per cent of all oöcytes retain their chromosomes of pair 3 intact, so that 37 per cent of the eggs are *cu*, 37 per cent *wo*. In the remaining 26 per cent the chromosomes break between *cu* and *wo* and exchange parts. Hence, 13 per cent of the eggs are *cu wo*, 13 per cent wild type (*Cu Wo*). When these eggs are fertilized by the *cu wo* spermatozoa, four kinds of offspring are produced, as illustrated in the lower part of Fig. 95. Naturally, these four kinds of offspring bear the same numerical relation to one another as did the four kinds of eggs, for they are the same individuals with identical spermatozoa added. Their ratio is thus 37:37:13:13.

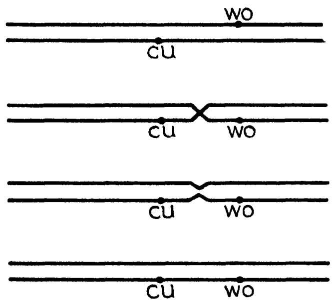


Fig. 96.—Diagram illustrating how two mutated genes in different chromosomes of the same pair may come to lie in the same chromosome, leaving wild-type genes at both loci in the other chromosome.

Proof of Linkage.—Though the order of events is that described in the preceding sections, linkage is discovered in concrete examples by reasoning backward. Chromosomes break, or remain intact, reduction division follows, different kinds of germ cells are produced and different offspring develop from them—all in the order named and described. However, one cannot watch all the cells, cannot see the chromosomes break and recombine, cannot tell by looking at them which genes they contain. All this has to be inferred from the adult offspring produced in crosses. For example, in Fig. 94, when the last cross results in only two kinds of offspring in equal numbers, it is known that the father (the only heterozygous parent) produced only two kinds of spermatozoa in equal numbers. Furthermore, from the characters of the offspring it is known that one of the kinds of spermatozoa had the genes *cu* and *wo*, the other kind their wild-type alleles. Since the chromosomes which came to this male from his parents contained *cu wo* and *Cu Wo*, respectively, it is obvious that these chromosomes have gone over to the germ cells without change. That is, there have been no breakage of the chromosomes and recombination of their parts.

In Fig. 95, when the last cross yields four kinds of offspring in the ratio of 37:37:13:13, it is known that the eggs of the mother (the heterozygous parent) produced four kinds of eggs likewise in the ratio of 37:37:13:13. The characters of the offspring also show what genes were in each of these kinds of eggs. They prove that the two more numerous classes had the same genes as did the respective parents of the heterozygous female. These two classes must therefore have received unbroken, unrecombined chromosomes from the reduction division. The two minority classes of eggs are similarly shown to contain chromosomes reconstituted through breakage and exchange of pieces. To explain the 37:37:13:13 ratio, it is necessary to assume that in 74 per cent of the oöcytes the chromosomes of pair 3 remain intact, at least they do not break between *cu* and *wo*, while in 26 per cent of the oöcytes such breakage and recombination occur.

The argument thus leads back from ratios of offspring and the characters they possess, to the behavior of the chromosomes necessary to explain those ratios and characters.

Multiple Crossing Over.—The breakage and recombination of parts of chromosomes are known as *crossing over*. It may happen at more than one place in the length of a chromosome pair. To detect the additional crossing over, it is necessary to have more heterozygous pairs of genes as markers. Specifically, the number of markers must be one more than the number of crossovers to be discovered. To prove double crossing over, therefore, the chromosomes must have unlike genes at each of three loci; to discover triple crossing over requires four loci with heterozygous genes.

To illustrate double crossing over concretely, take the characters black body (*b*), curved wing (*c*, different from curled, *cu*), and plexus or irregular wing veins (*px*), all in chromosome 2 in Drosophila. If a black curved plexus fly is crossed with the wild type, their offspring will have the composition shown in Fig. 97, *A*. When these F_1 flies produce their germ cells, oöcytes in which the chromosomes of this pair remain intact yield two kinds of eggs, *b c px* and wild type. If single crossing over occurs between *b* and *c* (Fig. 97, *B*), the resulting germ cells are *b* and *c px* (omitting mention of wild-type genes). Single crossing over between *c* and *px* yields eggs which are *b c* and *px*, respectively

(*C*). Finally, if there is double crossing over (*D*), the germ cells are *b px* and *c*.

Proof that these eight kinds of eggs are produced would be obtained by mating the F_1 females to males homozygous for all three recessives (*bb cc pxpx*). The offspring would show the various recessive characters for which there are genes in the several kinds of eggs in Fig. 97. The flies resulting from double crossover eggs would have curved wings, or they would be black bodied with plexus wing veins (omitting mention as usual of the wild-type characters).

Triple and quadruple crossovers have been obtained in Drosophila, but no new principle simple enough for elementary work would be illustrated by describing them.

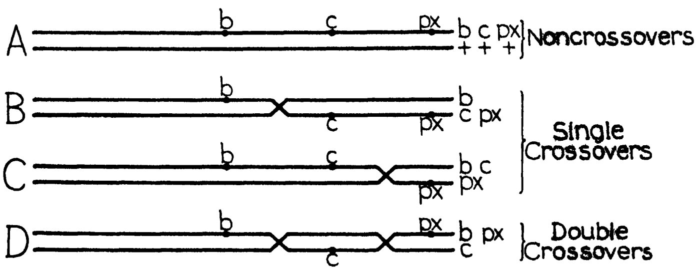


FIG. 97.—Single and double crossing over involving the genes for black body (*b*), curved wing (*c*), and plexus (*px*). The plus signs indicate wild-type genes, as does also the absence of any indicated mutated genes.

Crossing Over and Sex.—In Drosophila there is an important difference between the sexes with respect to linkage. While crossing over occurs in the female, there is under ordinary circumstances none in the male. In certain stocks of these flies it has been found possible to induce crossing over in the male by high temperature (Shull and Whittinghill 1934) and by X rays (Patterson and Suche 1934), and such exchange may happen in very rare individuals under ordinary conditions; but in typical experiments these possibilities may be ignored.

In at least one other organism, the silkworm moth, there is a similar difference between the sexes, but their relations are reversed. There is crossing over in the male but none in the female. The reason for this distinction between the sexes is unknown for either animal, but the reversal in the silkworm as compared to Drosophila is doubtless part of the reversed relation of sex to chromosomes in these species (pages 90–[illegible]).

In most organisms, however, both animals and plants, crossing over occurs in both sexes. Moreover, there is no striking difference in the frequency of that exchange in the two sexes. Unless it is specified in a particular example that it is Drosophila (a number of species) or the silkworm moth that is involved in linkage, crossing over should be assumed in either sex.

Chromosome Maps.—The genes *cu* and *wo* used to illustrate linkage were assigned definite locations in the third chromosome. How is it known where these genes are? They cannot be recognized in a microscope. Even in the greatly enlarged salivary gland chromosomes (Fig. 29), where perhaps the genes may be identified with the disks or crossbands, the tests which would indicate which of these disks are *cu* and *wo* have not yet been made. Knowledge of the position of genes comes from linkage experiments. It is assumed that crossing over (breakage of chromosomes) occurs at random throughout the length of the chromosomes. This is not a wholly correct assumption, for it is known that certain regions of a chromosome break somewhat more readily than others; but the supposed indiscriminate placement of the breakages may stand for this discussion.

Now, crossing over can be discovered by genetic experiments only when the chromosomes break at some point between two pairs of genes for which the individual is heterozygous. Breakage may occur at other places, but if there is not a heterozygous pair of genes on each side of this break to serve as markers, the crossover is not detected. If these pairs of heterozygous genes are far apart and the chromosome breakages occur at various points at random, many crossovers should occur between them and be discovered; if the pairs of genes are close together, recognized crossovers will be few. Under these circumstances, the distance between the marking genes is judged from the number of breakages between them. If breakage occurs between genes *a* and *b* in 10 per cent of the oöcytes, these genes are a certain distance apart; if breakage between *a* and *c* occurs in 20 per cent of the oöcytes, *a* and *c* are twice as far apart as *a* and *b* are.

The data just postulated permit a beginning of the mapping of the chromosome. Genes *a* and *b* are placed somewhere on the chromosome, at a distance from each other which is assumed to allow 10 per cent of crossing over (Fig. 98). Gene *c* must then

be placed twice that distance from *a*. There are, however, two positions which fulfill this requirement, one beyond *b* to the right, one to the left of *a*, as shown by the second and third lines of Fig. 98. Which of these is the correct position is determined by using *b* and *c* in a linkage experiment. If it is found that crossing over (breakage) between *b* and *c* occurs in about 10 per cent of the oöcytes, *c* is placed to the right; but if crossing over between *b* and *c* occurs in about 30 per cent of all oöcytes, *c* is placed 20 units to the left of *a*. The unit is a distance which permits 1 per cent of crossing over.

Let it be assumed that *c* is to the left of *a*. Another gene, *d*, is now used with one of the located genes, perhaps *b*, in a linkage experiment. If it is found to be 6 units from *b*, and in another experiment using *d* and *a* is found to be 4 units from *a*, gene *d* is

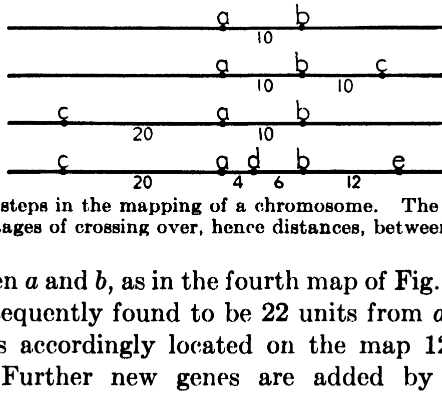


FIG. 98.—Early steps in the mapping of a chromosome. The numbers refer to the percentages of crossing over, hence distances, between the genes.

placed between *a* and *b*, as in the fourth map of Fig. 98. Suppose gene *e* is subsequently found to be 22 units from *a* and 42 units from *c*. It is accordingly located on the map 12 units to the right of *b*. Further new genes are added by testing them with two genes whose loci are already known, and placing them on the map at the place which will satisfy both distances thus determined.

After a considerable number of genes have thus been placed, one gene is found to occupy one extreme position beyond which no new gene has been located, another gene is located at the other end of the row. These genes are then assumed to be near the ends of the chromosomes, and one end is arbitrarily called "left," the other "right." These terms are mere conveniences, however. In making a map the earliest known gene is set down somewhere, and the next one known or located is put to the right of it, so that right and left depend merely on the order of discovery or testing. The gene that has held the extreme left

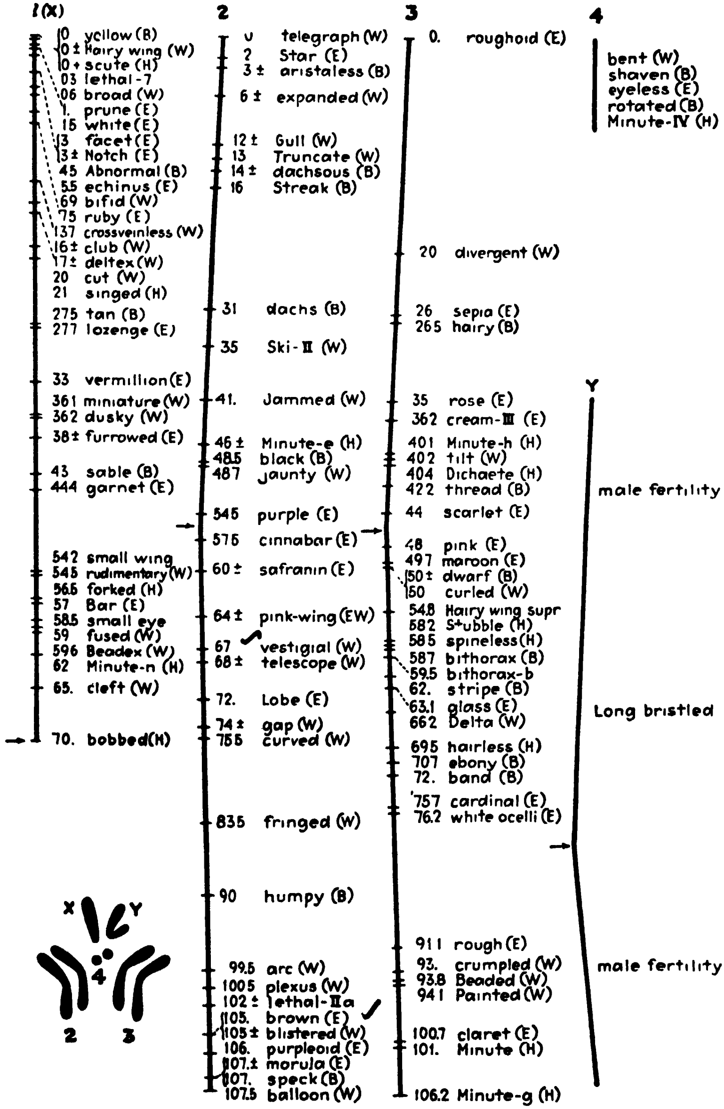


FIG. 99.—Partial maps of the chromosomes in *Drosophila melanogaster*. The letters in parentheses indicate the part of the body affected: *B*, body; *E*, eye; *H*, hairs; *W*, wings. Arrows point to attachment of spindle fibers. Locations in Y chromosome are not precisely known. Those in chromosome 4 are too closely linked for significant separation. (*From Sharp, Introduction to Cytology, McGraw-Hill Book Company, Inc., after Morgan, Sturtevant and Bridges, and Stern.*)

position for a long time is then assigned the locus 0, and others are given numbers which represent their ascertained distances from one another.

Maps showing a partial list of the genes located in the four chromosome pairs of Drosophila are shown in Fig. 99. The left end is at the top, right at the bottom. The gene for curled wing (*cu*), is at locus 50 of chromosome 3 and that for white ocelli (*wo*), is at locus 76.2. They are placed 26.2 units apart because crossing over occurs between them in 26.2 per cent of the oöcytes. From these maps, by noting the loci of any two genes, the amount of crossing over that might be expected to take place between them can be estimated. Thus, if vestigial wing, which is at locus 67 in chromosome 2, were tested with brown eye, whose locus is 2-105, there should be about 38 per cent of crossing over between them.

Linkage Ratios in F_2.—While testcrosses are the best *measures* of linkage, because the percentages of crossing over can be read off directly in the proportions of the different classes of offspring, linkage can usually be *detected* in F_2 generations because of the peculiar ratios which result. This is particularly true in Drosophila because of the absence of crossing over in the male, and it is true in other organisms if the linked genes are very close together. Some practice in determiming F_2 ratios in various linkage situations is desirable.

The genotype of a double heterozygote, if linkage is involved, must be written in such a way as to show which genes are in one chromosome, which in the other. If both dominant genes are in one chromosome, both recessives in the other, the formula is *CD-cd* or (*CD*)(*cd*). If there are one dominant and one recessive in each chromosome, the genotype is *Cd-cD* or (*Cd*)(*cD*). The F_2 ratio depends on which arrangement prevails.

Suppose the double heterozygote is *CD-cd*, and that there is 40 per cent of crossing over between the two pairs of genes. Assume further that the organism is Drosophila, with no crossing over in the male. The female *CD-cd* produces eggs of which 30 per cent are *CD*, 30 per cent *cd*, 20 per cent *Cd*, and 20 per cent *cD*. The male *CD-cd* produces only two kinds of spermatozoa of which 50 per cent are *CD*, and 50 per cent *cd*.

How these eggs and spermatozoa are combined may be ascertained by a calculation which resembles algebraic multiplication.

The percentages are converted into decimal fractions, and the multiplication problem is set down thus:

$$\begin{array}{l} .3\ CD + .3\ cd + .2\ Cd + .2\ cD \\ .5\ CD + .5\ cd \\ \hline \end{array}$$

The coefficients, indicating the proportions of the kinds of germ cells, are really multiplied. The formulas of the germ cells are not multiplied, but merely combined to form the genotype of one class of the offspring. In the offspring, the more usual formula may be used, not the one showing the arrangement of linked genes in the chromosomes, since the kinds of germ cells the offspring will produce are of no interest here.

The first partial product in this multiplication is .15 *CCDD*. The student will be allowed to carry out the remainder of the operation. When this is done, and the various partial products are collected into their respective phenotypes (appearances), it is found that the F_2 ratio is .65 CD:.15 cd:.10 Cd:.10 cD or 13:3:2:2. Were the genes independent, this ratio would be 9 CD:1 cd:3 Cd:3 cD. The existence of linkage is thus demonstrated by the observed ratio.

If the organism used is not Drosophila, so that there is crossing over in both sexes, the other assumptions made in the foregoing example would result in both eggs and spermatozoa 30 per cent *CD*, 30 per cent *cd*, 20 per cent *Cd*, and 20 per cent *cD*. The multiplication thus becomes:

$$\begin{array}{l} .3\ CD + .3\ cd + .2\ Cd + .2\ cD \\ .3\ CD + .3\ cd + .2\ Cd + .2\ cD \\ \hline \end{array}$$

If this multiplication is completed, and all of the same phenotype collected together, the F_2 ratio is found to be 59 CD:9 cd:16 Cd:16 cD. Again it is different from the 9:1:3:3 expected from independent genes, though not so strikingly different.

Next, assume that the linkage in the double heterozygote is *Cd-cD*, and that there is no crossing over in the male. The problem is:

$$\begin{array}{l} .3\ Cd + .3\ cD + .2\ CD + .2\ cd \\ .5\ Cd + .5\ cD \\ \hline \end{array}$$

From this the phenotypic ratio in F_2 is 2 CD:1 Cd:1 cD. There is no cd class at all—a very easily detected indication of linkage.

Finally, let the linkage arrangement be *Cd-cD*, and assume that as in most species there is crossing over in the male. The multiplication is:

$$\begin{array}{c} .3\ Cd + .3\ cD + .2\ CD + .2\ cd \\ .3\ Cd + .3\ cD + .2\ CD + .2\ cd \\ \hline \end{array}$$

The completed product, arranged into its phenotypes, is in the ratio 54 CD:21 Cd:21 cD:4 cd. Perhaps this would not be recognized as a result of linkage, since it might pass for 9:3:3:1.

From most of the ratios described in this section there is no *simple* or *direct* way of determining what the percentage of crossing over is. One of them gives absolutely no clue to that percentage. For species in which there is equal crossing over in both sexes, there are statistical methods which enable one to ascertain the amount of crossing over (Fisher and Balmukand 1928; Wiener 1932), and tables have been prepared by means of which rough estimates of the degree of linkage may be quickly made. These methods are scarcely suitable, however, for elementary work.

Number of Linkage Groups.—The characters of any organism which are linked with each other because their genes are all in one pair of chromosomes constitute what is called a linkage group. Any two or more characters within one such group are linked; any two or more characters all of different linkage groups are independent in distribution.

The number of linkage groups in any animal or plant is the number of chromosome pairs. Only in a species which is genetically very well understood could characters of all of these groups be known. Drosophila is the only animal of which this is true; the commonest species has four pairs of chromosomes, and four linkage groups have long been on record. Corn is the only plant so well known; it has 10 pairs of chromosomes and 10 known linkage groups.

Other organisms are less well known. For these others no distinction will be made between linkage *groups* (two or more linked characters) and single independent characters. In rats, for example, only 2 groups of linked characters are known, but 5 other chromosomes are marked by single independent

genes (Blunn and Gregory 1937). The precise number of chromosomes in rats is unknown but much larger than 7. Likewise, in pigeons only two linkage groups have been discovered, but several additional chromosomes have single known genes in them (Hollander 1936); the number of chromosomes is considerably greater. In tomatoes 10 of the 12 chromosomes have had characters assigned to them (MacArthur 1934); in the morning-glory 12 out of 15 (Imai 1933); in the mouse 15 out of 20 (Castle 1935); in the rabbit 11 out of 22 (Castle 1934); and in poultry 10 out of the 16 or 17 which fowls must possess (Warren 1938).

Mechanism of Crossing Over.—The diagrams in Figs. 96 and 97 may lead to the impression that crossing over is due to some form of twisting or crossing of the chromosomes, in which the chromosomes come into contact and then pull apart without untwisting. That is a conceivable method and was long the prevalent theory regarding the mechanism of crossing over. Plenty of chromosomes in various organisms show this spiral twisting (Fig. 100) or cross formation, giving at least plausibility to the old theory. It is now being maintained, however, that the crosses seen in paired chromosomes are results, not causes, of crossing over—that the chromosomes already have exchanged pieces and that the appearance of crossing or twisting is due to the new arrangement of the parts. How the pieces are brought into their new connections is not known; the physiology of chromosome pairing is in need of elucidation.

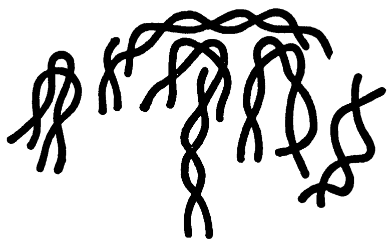


Fig. 100.—Twisting of pairs of homologous chromosomes in Batracoseps, foundation of one theory of the mechanism of crossing over. (*After Janssens in La Cellule.*)

The time of crossing over must be earlier than the reduction division. If it happens immediately before reduction, the two crossover classes should be equally numerous. Ordinarily their numbers are approximately equal, as is shown to be true of the wild-type and *cu wo* germ cells in Fig. 95. Occasionally, however, the chromosomal exchange occurs some time before reduction. If, after such early crossing over, one of the resulting cells divides several times before the reduction division, while the other cell does not divide, the two classes of crossovers are very unequal in

number. Unequally numerous crossover classes are regularly attributed to early crossing over and additional divisions of one of the reconstituted cells.

Prior to reduction the chromosomes of a pair have come together side by side. In many organisms each of these chromosomes has at the same time become duplicated. A quadruple chromosomal body is thus formed in which there are two sister chromatids derived from the maternal chromosome, two other sister chromatids coming from the paternal chromosome. Crossing over must often take place during this four-strand stage. While exchange might conceivably occur between any two of these chromatids, the bulk of the evidence indicates that sister strands do not cross over with each other, but that always a maternal and a paternal chromatid are involved. Furthermore, at any given level in the four-strand body, only two chromatids cross over. The others may cross over at some other point in the length of the chromosome, however.

Search for Linkage in Man.—There can be little doubt that linkage occurs in man just as in other animals. So far the only indubitable instance of it is the group of sex-linked characters- red-green color blindness, hemophilia, probably Leber's optic nerve atrophy, Gower's muscular paralysis, some night blindness, some myopia, and others. Since their genes are all in the X chromosome, they must be linked with one another. This one linkage group is known only because the relation to sex is so easy to discover. No family pedigree is yet on record which involves any two of these characters in such a way as to show that they are linked. Madlener (1928) did indeed find both hemophilia and color blindness in one family, and the facts are in harmony with their linkage with each other; but the number of individuals is too small to prove them linked without recourse to the argument that both are sex-linked. Davenport (1930) searched for family histories in which two or more sex-linked characters exist, for the purpose of discovering whether they showed direct signs of linkage, but his results were not very conclusive. Yet it may be confidently assumed that, were two such characters present in a family large enough to give evidence of linkage, the linkage of the two characters would be proved.

Autosomal linkage cannot be detected in this indirect way. Consequently, no example has been proved. The reasons are

plain. First, man has 24 pairs of chromosomes, hence on the average each linkage group includes only about 4 per cent of his hereditary characters, as against more than 30 per cent in some of the groups in Drosophila. Second, human families are small. Linkage is proved by mating a double heterozygote (phenotypically a double dominant) to a double recessive and getting progeny of four classes, two of which are about equal and more numerous than the other two which are likewise nearly equal. No human family is large enough to make such ratios secure. Some very suggestive families have been found, such as the one comprising 16 children, some of whom had dark hair and crooked little fingers, the rest blond hair and normal fingers. Yet even this could happen by accident to two independent characters. Could the same peculiar relation between these same characters be found in another family of some size, the case for linkage would be much better.

Other methods of detecting linkage have been proposed, but they involve statistical methods and are not suitable for elementary presentation. Reference has already been made to methods developed by Fisher and Balmukand (1928) and by Wiener (1932). These methods render linkage probable when it exists, but adequate applications of them to examples which prove linkage have yet to be made. Some such tests have, however, shown certain characters to be independent. Wiener's own work indicated that the agglutinogens M and N are independent of A and B (pages 108–112).

A word of caution is needed against the assumption that two characters found together most of the time are linked. Linkage does not make two characters hang together *in the long run*. It does so in some single families in which their genes happened to start together in the same chromosome; but in other single families linkage keeps these characters apart most of the time, because their genes started in opposite chromosomes of a pair. On the whole, therefore, linked characters are neither more nor less commonly together than independent characters are. When two qualities are found to occur together more frequently than would be expected from random distribution, the probable explanation is that they have some part of their genetic (physiological) basis in common. This relation could be called correlation (see Appendix), but it is not linkage.

CHAPTER XIX

PROOF THAT GENES ARE IN CHROMOSOMES

From the first description of the genetic mechanism in the early chapters and in all the explanations of breeding experiments, it was stated that the genes are in the chromosomes. It would have been difficult to offer proof of the correctness of this statement when it was first made, though the fact that so many events appear to have a reasonable and harmonious foundation if the genes are in the chromosomes should create a presumption in its favor. It is time now to assemble a few of the more pertinent facts which go to show that the mechanism of heredity has been correctly portrayed.

The Mendelian Mechanism.—Among the more general evidences that the chromosomes are the conveyors of the genes is the correspondence between the behavior of the genes and that of the chromosomes. Breeding experiments show as a logical necessity that whatever an organism receives from its two parents, with respect to a certain characteristic, it passes on *separately* to *different* individuals among its offspring. Calling the things it receives genes, no matter what or where they are, one must conclude from crosses of unlike parents that the genes contributed by the parents are separated—segregated, we say—in the germ cells of the next generation, so that each germ cell receives one of them, not both. F_2 generations and back-crosses would not be what they are without the segregation of the genes of a pair into different germ cells. Now, all this has an explanation if the genes are in the chromosomes, for the reduction division can then be the cause of segregation.

When two characters are studied simultaneously, it often happens that these characters prove to be independent of each other in their distribution to the offspring. The ratios of offspring in F_2 and other hybrid generations demand this independence. The ratios would be different if the genes were not independent. Here again there is a reasonable explanation of the logically

deduced relation if the genes are in the chromosomes; for the different pairs of chromosomes are ordinarily free from any mutual interference.

Even when the two pairs of genes are not independent, as is true in all instances of linkage, the chromosomes offer an excellent explanation if it be assumed that both pairs of genes are in the same pair of chromosomes, for the chromosomes provide the means of preventing independence.

No normal behavior of genes has yet been discovered from hybridization experiments which is not paralleled by normal behavior of chromosomes. This is a circumstantial indication that the two are connected.

Haploid and Diploid Organisms.—Any organism which is derived from two parents should have two homologous chromosomes of each kind. Likewise any organism with two parents should have two genes of each kind. It has been assumed that any two homologous genes are in two homologous chromosomes. If, now, any individual of such a species is haploid, whether as a normal phase of the cycle or as an accident, it should have only one gene of each kind—if the genes are in the chromosomes.

In the simple plants, or algae, the only diploid cells ever produced are the zygotes formed by the fusion of two cells in sexual reproduction. Such a zygote, by two divisions, produces four cells which may be observed to be haploid. Reduction has occurred in one of the divisions, and the cytological observations go to show that it is the first of the two divisions in which the homologous chromosomes separate. Now, what happens to the genes in the course of these divisions? In one species of alga, two varieties having a different shape of eye spot were crossed, and the zygote was observed to have an eye spot of intermediate form. This shows that the zygote had two genes for eye shape, and neither was dominant over the other. When, however, the zygote had divided into the four characteristic vegetative cells, two of these cells had eye spots of one of the parental shapes, the remaining two of the other parental shape. None of these cells had intermediate eye spots. These facts show that each of the four cells had but one gene for eye shape, while the zygote had two genes. The diploid cell had two genes, haploid cells only one. This should be the situation if the genes are in the chromosomes.

The cycle of the mosses includes both a haploid and a diploid phase, which are represented by conspicuous structures. From a spore (Fig. 36, *a*) there is produced a group of cellular threads in the soil. From these threads, in turn, develops the moss plant, or gametophyte (Fig. 36, *b*). At the top of the gametophyte, eggs and spermatozoids (male cells) are formed. Everything so far described is haploid. There is no reduction in the number of chromosomes when the germ cells are formed, for the gametophyte itself is already haploid. When fertilization of an egg by a spermatozoid takes place, the combined cell is diploid. From this fertilized egg develops the sporophyte (*c*), which remains attached to the top of the gametophyte. The sporophyte, like the fertilized egg from which it springs, is diploid. Then, within the capsule of the sporophyte, spores are formed. These are haploid. When, therefore, a cell in the sporophyte divides twice to form four spores, reduction of the chromosomes takes place. With the haploid spores, the cycle may begin all over again.

What is the number of genes in these two generations of the moss cycle? Fortunately, there are enough variations in the mosses to answer this question. As for the diploid sporophyte generations, there are different colors, red, green, orange, etc. Each race, when grown by itself, produces sporophytes of its own peculiar color. When, however, two of them are crossed, by controlled fertilization of the egg by a spermatozoid from another race, the sporophyte which develops from the egg is intermediate in color. This color is due to lack of dominance, and could occur only if two different genes were present. The diploid sporophyte therefore has two genes for color in each cell.

In the gametophyte, the shape of the leaves is different in different varieties. The genes for leaf shape go into the eggs and spermatozoids, and when two varieties are crossed, two different genes enter the sporophyte. In the sporophyte, which has no leaves, these genes cannot come to expression; but they go into the spores, from them to the cellular threads in the soil, and finally into the gametophytes where they can come to expression. Here, however, the leaves are not intermediate; in some individual gametophytes the leaves have one of the varietal shapes, in other individuals the other shape. That the leaves would be intermediate if both genes were together in a gametophyte is shown by regenerating a gametophyte from a heterozygous

sporophyte; but this is a phase of the work that must be passed over with this brief reference. All this means that each gametophyte has only the one gene or the other, not both of them. It is thus shown that the haploid generation has only one gene for a character, not two as the diploid generation has.

Number of Linkage Groups.—It was shown in the preceding chapter that in no organism does the number of known groups of linked characters exceed the number of pairs of chromosomes. If the genes were not in the chromosomes, that is, if linkage were due to something else than the chromosomes, it would presumably be possible to have more linkage groups than there are chromosome pairs. This is negative evidence, but important, since if the linkage groups exceeded the chromosomes in number, the genes could hardly be regarded as being in the chromosomes, or some peculiar chromosome behavior (fragmentation, perhaps) would have to be sought.

Sex-linked Genes.—In Chap. X it was shown that genes for sex-linked characters are possessed unequally by the two sexes. In man and the mammals generally, and in most insects, the female must have two genes for such characters, the male only one. In no other way can the peculiar type of inheritance of sex-linked characters be explained. The only anatomical feature in which the sexes can be observed to differ in precisely this way is the X chromosome; the female has two X chromosomes, the male only one. The assumption that the sex-linked genes are in this chromosome is almost necessary.

Closely related to sex-linkage is Y-chromosome inheritance. Some characters pass only from father to son. When these are known not to be secondary sex characters, because not all males have them, it is necessary to find some structure that passes only from father to son, if an explanation is to be had. The Y chromosome is the only known feature of mammals and most insects which is known to be transmitted in this way, and it is natural to suppose that the genes in question are in the Y.

Nondisjunction.—Sometimes a genetic experiment shows that a certain individual has only one gene of a given pair, although it has the expected two genes of other pairs. The absence of one gene is shown in a variety of ways. It may appear that a gene of that pair is going to only half of the offspring, after the manner of sex-linked genes in a male fly. Or a recessive gene may come to expression in a heterozygote, which could happen

only if the dominant gene of the same pair, supposedly present, were absent or inactivated. These peculiarities are characteristic of the haplo-4 Drosophila described on page 114, and this particular fly lacks one of the small fourth chromosomes (Fig. 63). The obvious explanation is that the gene that is going into only half the offspring, or that is coming to expression in a supposed heterozygote, is in the one fourth chromosome which remains. Any instance of nondisjunction may bring forth similar modifications of inheritance.

Duplicate Genes.—By means of a 15:1 ratio in F_2 and confirmatory evidence from the F_3 generation, it has several times been shown that a species has four genes for a certain character, instead of the usual two. These two pairs are precisely alike and hence are known as duplicate genes. Examples are known in the shape of seed capsule in shepherd's-purse (page 158), in the red pericarp color of rice (Ramiah and Mudaliar 1935), and others. In some such examples the species is known, from observation of its chromosomes, to be tetraploid. This merely means that it has twice as many chromosomes as does some other species which is regarded as diploid. It is believed that the tetraploid species was derived from the diploid by a duplication of the chromosomes, which is a not exactly rare way of producing new species in plants. If it may be assumed that the genes are in the chromosomes, the duplication explains not only the origin of a species but the existence of duplicate genes as well.

Chromosome Aberrations.—Occasionally in such a genetically well-known organism as Drosophila or corn, a gene is found to change its linkage relations. A character which has long been known to be a member of linkage group 2, for example, is suddenly found linked with group 3 instead. At the same time, some individuals may show unexpected peculiarities in the group 2 characters—a recessive character of group 2 may show in a heterozygote, or the amount of crossing over between genes in a certain part of group 2 may be greatly reduced, even to zero. In some of these instances, it has been possible, by examining the chromosomes of these irregular individuals, particularly those which have the second-group characters tied up with group 3, to observe that a piece of chromosome 2 has been broken off and attached somewhere to chromosome 3. A transfer of a fragment of a chromosome to some other *nonhomologous* chromosome is called a *translocation*. If it may be assumed that the

genes which are behaving unusually are in the translocated fragment, all the peculiarities described above are explained.

Comparable disturbances are produced when a piece of a chromosome is broken away and attached to some other whole *homologous* chromosome, as for example, a piece of chromosome 2 attached to a whole chromosome 2. This type of change is known as a *duplication*, from the fact that the elements in the transferred piece now occur twice in the receiving chromosome. Somewhat different irregularities follow when a segment of a chromosome is taken out, turned end for end, and put back in again. This is not quite the way in which the reversal happens, but the end result is correctly described. This latter aberration is known as an *inversion*. For both duplications and inversions, the corresponding behavior of certain genes has been detected by crossing experiments, and the chromosome irregularities later observed in the cells with a microscope.

Salivary Gland Chromosomes.—With the discovery of the enormous chromosomes of the salivary glands in Drosophila (Fig. 29), translocations, duplications, inversions, and deficiencies (vacant places left when a chromosome fragment has been removed) have assumed a new importance. Whenever a character shows any irregularity in its behavior, in any stock of flies, salivary glands from larvae of that stock may be examined with some hope of observing exactly what has happened. When genetic experiments show that certain genes are missing from linkage group 2, for example, salivary gland chromosome 2 may be seen to lack certain of its disks. Many disks may be missing, though the experiment detected only the absence of one gene. Later another deficiency involving the same gene may be found to accompany the absence of a certain other series of disks in the salivary gland chromosome. The missing segments in these two examples overlap, however, and it is thus known that the gene which both experiments indicated was missing must be in the overlapping part. By subsequent similar tests with the same gene, the overlapping portions may be reduced to a very small amount. In this manner, it has been possible to say that a certain gene is located in the region of two or three definite disks.

These and other related phenomena seem to offer abundant confirmation of the view that the genes are in the chromosomes. There is among geneticists no difference of opinion on this question.

CHAPTER XX

NON-MENDELIAN INHERITANCE

The evidence that the genes are in the chromosomes completely identifies these nuclear bodies with the mechanism of Mendelian heredity. Since there is, however, a group of phenomena that do not follow the Mendelian rules, it is necessary to seek supplementary vehicles to explain them in order to complete the sketch of the genetic equipment. Before we turn to these nonconformist phenomena, the rules which they violate must be clearly in mind. What is Mendelian heredity?

Mendelian Heredity Defined.—Mendel's laws, as derived from his own experiments, involved (1) segregation of the genes of the same pair, and (2) independent assortment of genes of different pairs. Since Mendel's time, it has been found that the second of these, independent assortment, is not universal. Linkage has been found to be exceedingly common. Mendel never observed linkage; should linkage then be excluded from Mendelian heredity? Considering that linkage is due to the very things (chromosomes) which are responsible for independent assortment, geneticists chose not to separate linkage from the other fundamental features of heredity and have extended the term Mendelian heredity to cover it.

Once this extension was accepted, it was logical to include the other things which are dependent on the regular behavior of chromosomes, such as sex-linked inheritance. And finally, even those phenomena which result from irregularities of chromosome conduct—nondisjunction, translocation, duplication, deficiency—were regarded as Mendelian phenomena. At the present time, any transmission which is directly dependent on chromosomes is considered Mendelian heredity.

The inheritance which may be said to disregard Mendelian rules must therefore depend on something else than chromosomes. Most of the phenomena of this exceptional sort relate to inequality of the influence of male and female parents. Some of

these inequalities are permanent, others gradually decrease and disappear.

Variegation in Plants.—One of the permanent inequalities between the two parents is their relation to variegation in plants. Green plants owe their color to chlorophyll-bearing bodies, the *plastids*, in their cells (Fig. 5). So long as the plastids are normal healthy organs, the green color is maintained. Sometimes, however, they are deficient in their power to produce chlorophyll; such plastids become pale green, or yellow, or white. If the deficiency extends throughout the plant, it is fatal, because nutrition depends on the photosynthesis for which normal chlorophyll is a prerequisite. Only young plants, therefore, which are still drawing nourishment from the seed, can afford to be white. Older plants may, however, be yellow or white in spots, a condition known as variegation.

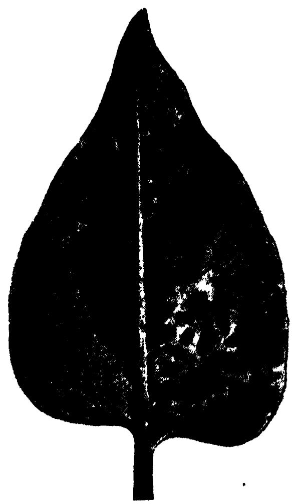


FIG. 101.—Variegated leaf of the four-o'clock *Mirabilis jalapa*. (*After Correns.*)

When a variegated four-o'clock (*Mirabilis jalapa*, Fig. 101) is crossed with a solid green one, the offspring are always like the mother. If the female parent was variegated, the offspring are variegated; if the mother was solid green, the offspring are green. On a variegated plant, some branches may be solid green, some solid white, some variegated. When a flower on a green branch and one on a white branch are cross-pollinated, the offspring are again like the mother, whichever way the cross was made.

Corn also has its varieties with pale green stripes among the dark green of the leaves (Fig. 102). The variety illustrated arose suddenly in a single plant among more than a hundred in one family. This one plant was raised, and produced one ear. The grains on this ear were planted the next year in rows in a plat, in the order in which they were placed on the cob. The majority of the plants growing from these seeds were green, but one large area of the original ear yielded pale plants (Fig. 103) which soon died. But along the margins of this white area,

the grains produced many variegated plants. From these originated a variety which continued to produce striped plants. Any crosses between striped and green plants yielded only striped or only green, to agree with the mother.

Similar inheritance only from the mother is found in Chinese plantain lilies (Yasui 1929), in a pale green variety of barley (Robertson 1937), and in a tricolored barley (Imai 1936).

In every example the explanation is that the plastids are carried over from generation to generation only in the egg. Recognizable plastids are not to be seen in either egg or pollen, but some representative of these structures must be present in the egg. Even if similar representatives were in the pollen, they might never be transmitted to the offspring, for what reaches the egg from the pollen tube in fertilization (pages 39 and 65) is practically only a male generative nucleus. If the plastids reside in the cytoplasm (protoplasm outside the nucleus) of the egg, and not in the pollen or are ineffective in the pollen, the strictly uniparental transmission of variegation is explained. This type of inheritance goes along undiminished through any number of generations.

FIG. 102.—Variegated corn; stripes of pale green in otherwise green leaves. This variegation is transmitted only by the mother. (*Photograph by E. G. Anderson.*)

Male Sterility in Corn.—From Peru comes a strain of corn in which the pollen is largely or completely sterile (Rhoades 1931). When this variety was crossed with North American lines with fertile pollen, mutations of the latter known to reside in particular chromosomes were used as markers. It was found in this way that substituting any of the chromosomes of the pollen-fertile North American strains for the homologous ones of the Peruvian strain did not remove the sterility of the latter. Nine of the ten linkage groups (depending on chromosomes)

were found free of any gene for sterility, while the tenth group was not fully tested. Pollen from those plants that were not completely pollen-sterile was used in crosses and was found not to carry any gene for sterility. Moreover, the maturation divisions of the reproductive cells were normal, hence sterility could not be due to irregular distribution of chromosomes.

FIG. 103. Plat of corn seedlings from striped parent. The seeds were planted in the same relative positions as they occupied in the ear. The plat shows that seeds from one considerable area on the ear produced mostly pale green plants. Along the border of this area the seeds produced many striped plants. (*From Anderson in Botanical Gazette.*)

It was concluded that something outside the chromosomes must be responsible for the sterility, and this condition was handed down indefinitely from generation to generation.

Breast Cancer in Mice.—Similarly permanent, though not necessarily to the full degree of the original ancestor, appears to be the maternal transmission of susceptibility to breast cancer in mice (Little 1936). Strains of mice differ in their liability to this disease. In a "high" line as many as 85 or even 100 per cent of the individuals develop the tumors, though any line exhibiting 50 per cent incidence is regarded as high. In "low" lines, few individuals (down to 0.1 per cent) are susceptible. Crossing a

high female with a low male yielded 39 per cent cancerous offspring, while in the reciprocal cross (low female by high male) only 6 per cent of the offspring developed breast cancer.

When the high F_1 females are used in similar crosses, the same preponderant influence of the mother is seen. This maternal transmission is said to follow the female line indefinitely. It is suggested that the real reason in this instance may be, not heredity, but some influence of the milk.

Cytoplasmic Influence under Control of Genes.—Some maternal influences, exerted through the cytoplasm of the cells of the female, are nevertheless controlled by genes. An

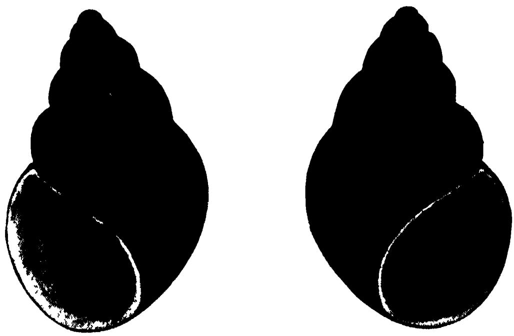


FIG. 104.—Sinistral (left) and dextral shells of snail. The direction of the coiling appears to be determined by the hereditary nature, not of the snail itself, but of its mother.

example is the inheritance of direction of coiling in snail shells. In some species the shell always coils to the right (dextral), in other species always to the left (sinistral). In a few kinds both dextral and sinistral individuals are found (Fig. 104), and in one of these it has been shown that dextral coiling is dominant over sinistral.

The direction of coiling, however, is not determined by the genes of the snail itself, but by those of its mother. If the gene for sinistral coiling is symbolized by l and that for dextral by L, any female whose genotype is LL or Ll will produce only dextral offspring. Even if she is mated with a male which is Ll or ll, and some of their offspring are ll, yet these homozygous recessives will show the dominant character because their mother carried the dominant gene L.

The reason for this peculiarity is that the cytoplasm of the eggs has had impressed on it, before the reduction division, the

influence which governs direction of coiling. Any female carrying *L* will produce only eggs that have experienced this influence, even if some of them have the gene *l* after maturation. Any *ll* offspring, though themselves dextral, will produce in their turn only sinistral offspring, however, for the cytoplasm of *their* eggs grows under the guidance of the sinistral gene.

How plausible this maternal influence is will be understood from the fact that direction of coiling in a snail is determined by the position which the spindles of the dividing cells take in the early cleavages of the egg. The cytoplasm governs this position, hence controls direction of coiling.

Possibly a similar situation exists in the eggs of the silkworm moth. The color of the shell of these eggs may be green or white; which color any egg has depends on the genes of the mother, not on those of the mature egg itself. Green is dominant over white, so that if the female has one gene for green, all her eggs will be green, even though some of them contain only the white gene after reduction. Whether the inheritance of egg shell color is like that of coiling in snails depends on what produces the shell. If the egg itself secretes it, then the cytoplasm of the egg must have had impressed on it, before the reduction division, whatever influence decides shell color, and the situation is identical with that of the snails. If, however, the egg shell is produced by the oviduct or some other tissue of the mother, we are merely witnessing a maternal character which is being borne by an early stage of the offspring. This would not be cytoplasmic (non-Mendelian) inheritance, because the shell would be developed under the direction of maternal genes (in the oviduct cells) as well as maternal cytoplasm.

Cytoplasmic influence is likewise shown in the eye color of the meal moth Ephestia, though it is transitory and partial. When a heterozygous female (*Aa*) is mated with a recessive male (*aa*), some of the offspring are of course homozygous recessives (*aa*). These larvae show the dominant eye color at first (a cytoplasmic holdover from the mother) but become intermediate later (Caspari 1936).

Diminution of Cytoplasmic Effect in Subsequent Generations. The disappearance of the cytoplasmic effect is more leisurely in some organisms and requires several generations. In beans, different varieties have very unequal resistance to the mosaic

disease, which is caused by a virus. Both Michigan Robust and Corbett Refugee are resistant varieties (Parker 1936). When they are crossed with susceptible kinds, the F_1 plants resemble the mother much more than the father. If Michigan Robust is pollinated from a susceptible plant, the F_1 generation is resistant, though somewhat less so than Robust itself. If a susceptible variety is pollinated from either Robust or Refugee, the F_1 plants are susceptible. The resistance shown by F_1 from the former cross is continued, but diminished, in F_2; and there is still some extra resistance in F_3. Under the influence of genes which should make these later generations intermediate, the cytoplasm thus gradually loses its high resistance.

Incidentally, it may be pointed out that the basis of resistance is not the same in all resistant varieties, for, when Michigan Robust and Corbett Refugee are crossed (F_1 being resistant), some susceptibility crops up in F_2. This should not occur if the feature responsible for resistance were the same in both varieties.

Rate of growth in young mice is found to depend more on the mother than on the father, and the maternal influence continues as far as the F_2 generation (Marshak 1936). In Drosophila heating the larvae causes an increase in the number of mutations produced in their germ cells, and some of this increase holds over to the next generation, long after the heat treatment ceased. This postponed induction of mutations has been attributed to an effect of the heat on the cytoplasm, which then continues to cause mutation of the genes. The effect, whatever it is, is temporary, however, for it gradually disappears (Jollos 1935) through about six successive hybrid generations.

Mechanisms of Non-Mendelian Heredity.—All the examples of transmission which does not conform to chromosome behavior are brought to notice by some difference in the influence of the two parents on the offspring. Usually it is a preponderance of maternal control. The mechanism responsible for this difference is clear in characters affecting the plastid colors of plants, for the plastids themselves may be transmitted through the cytoplasm of the egg but not through the pollen.

In species in which sex is dependent on Z and W chromosomes (page 91), the W chromosome passes only from mother to daughter and might conceivably enable the female (ZW) to exert influences of which the male (ZZ) is incapable. Goldschmidt debated long the question whether, in his theory of sex

determination in the gypsy moth, the female-determining influence should be attributed to the W chromosome or to the cytoplasm of the egg. His final conclusion (Goldschmidt 1934) is that it is the cytoplasm.

What part of the cytoplasm could be held responsible for maternal influence is uncertain. Any structure charged with this task would have to be of rather general occurrence. So far the *mitochondria* (Fig. 5) are the only objects (other than plastids) suggested for the non-Mendelian role, but there is no good evidence that they ever play it.

Cytoplasmic transmission is not to be confused with the greater influence of the female due to the source of embryonic nutrition in mammals. The mammalian fetus is connected with the mother's uterus by means of the umbilical cord and placenta and receives all its nutrition by diffusion from the mother's blood. Characters that are modifiable by nutrition may show a greater influence of the female parent and could lead to the impression that non-Mendelian heredity is at work. Castle *et al.* (1936) found, for example, that in rabbits offspring from large-race mothers crossed with small-race fathers are larger than offspring of the reciprocal cross. The difference is not great but is statistically significant and could be due either to cytoplasmic transmission or to influence of the mother during gestation.

Non-Mendelian Heredity in Man.—Established instances of cytoplasmic inheritance are scarcely to be expected in man, but they are as probable in the human race as in other mammals. The only suggestion that a human character is cytoplasmic appears to be that of Imai and Moriwaki (1936) regarding Leber's disease. This disease, which is atrophy of the optic nerve, is not very common, and about a quarter of the 200 known examples are Japanese. The mode of inheritance has been generally regarded as sex-linked, but some investigators have recognized a difference between European family histories and those from Japan. The latter have seemed to some to rest on an autosomal dominant or even on two autosomal dominants. The suggestion of Imai and Moriwaki is that the disease is cytoplasmically transmitted, but that it is differently affected by the male and female hormones. Its greater prevalence in males, which led to the concept that it is sex-linked, is held by them to be quite as well explained by the differential effect of sex hormones.

CHAPTER XXI

DETERMINATION AND DEVELOPMENT OF SEX

The dependence of sex on chromosomes was pointed out in Chap. X in explanation of sex-linked characters and again in Chap. XIX as proof that the genes are in the chromosomes. In the mammals, most insects, and some fishes there are two X chromosomes in the female and only one in the male. Along with the one X of the male there may or may not be a Y chromosome. In the moths, birds, some fishes, and the caddis flies, the chromosome relation to sex is reversed. The male has two similar chromosomes related to sex (here called Z chromosomes), while the female has only one. Along with the one Z of the female there may or may not be a W chromosome. As previously indicated, the X and Y, Z and W chromosomes are known as heterosomes.

Heterosomes in Maturation and Fertilization.—The manipulation of these chromosomes in maturation is illustrated for an XO species—one lacking a Y chromosome—in Fig. 105, which shows the male cells of the bug Protenor. The male has 13 chromosomes, made up of 12 autosomes and one X chromosome. At the left of the figure is the reduction division, with the single X going to the lower pole. At the completion of this division the two daughter cells, shown in the middle of the figure, are unlike in their chromosomes; the one at the top has 6 autosomes, the lower one 6 autosomes plus an X chromosome. The final spermatozoa are likewise of these same two kinds. The eggs of the female of this species all have 6 autosomes plus an X, like the lower male cell. When these eggs are fertilized by the two kinds of spermatozoa, there result the two different combinations shown at the right in Fig. 105. One combination consists of 12 autosomes and one X (above), and this kind develops into males. The other combination (below) has 12 autosomes and two X chromosomes, and this kind develops into females. The spermatozoa are thus the sex-determining agents, the X-bearing

type being female-determining, the one without an X (whether with a Y or not) being male determining.

In the moths, birds, and other groups having the Z or Z and W chromosomes, it is the female which produces two kinds of germ cells. Some of her eggs contain a Z chromosome; others do not. All spermatozoa are alike in containing a Z. Here the eggs are sex-determining. An egg with a Z chromosome is male-determining, one without a Z (whether with a W or not) is female-determining.

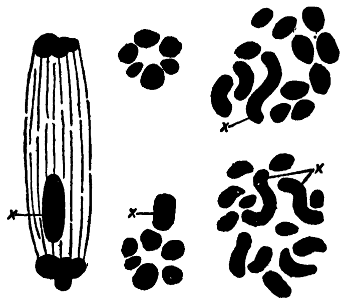


FIG. 105.—Chromosomes of an XO male, the bug Protenor. Reduction division at left, resulting cells in middle. At right, diploid condition restored by union of the two kinds of spermatozoa with the one kind (6 + X) of egg. *x*, the X chromosome. (*Modified from Wilson*)

Y chromosomes when present pass down the male line exclusively, in normal reproduction, but Y is not male-determining. The presence of but one X is what makes the individual male. W chromosomes are transmitted only in the female line but are not female-determining. It is the presence of but one Z which makes the individual a female. Both X and Z chromosomes act in relation to other chromosomes, or in relation to the cytoplasm, as is pointed out later.

Sex and Chromosomes in Plants.—Sex is not so sharp a distinction in plants as in animals, partly because many plants are hermaphroditic, partly because sex is not always expressed in structures. The problem of determination is somewhat different also, because of the separation of gametophyte and sporophyte generations, described for the mosses on pages 65 and 202. In the flowering plants the gametophyte is reduced to a structure composed of 3 to 8 cells; the male gametophyte is the pollen grain and is formed in an anther; the female gametophyte is the embryo sac (page 39) and is lodged in a carpel. It is the gametophytes which really possess sex. If a sporophyte generation, which is the conspicuous individual in flowering plants, allows only one kind of gametophyte to develop within it, these sporophytes are then more or less properly designated as male or female.

Sex chromosomes were first discovered in plants by Allen, studying a liverwort. Male gametophytes of this liverwort were found to contain 7 autosomes and a Y chromosome, female gametophytes 7 autosomes and an X chromosome. The sperm and eggs produced by these gametophytes experienced no further reduction, just as in mosses, and were of the same chromosomal constitution as the respective gametophytes. The fertilized egg thus contained 14 autosomes and an X and a Y. The sporophyte developing from the fertilized egg was likewise 14A + X + Y and was, of course, sexless. The Y chromosome in these plants is much smaller than the X.

In the flowering plants also a number of species with distinguishable chromosomes associated with sex have been found. Female plants of Melandrium (related to the pinks), for example, have two large X chromosomes, male plants one X and a smaller Y. Allen, reviewing this subject in 1932, reported that 45 species of flowering plants, belonging to 19 genera, had been found to possess X and Y chromosomes. Mostly it is the male which is heterozygous (XY), but in certain strawberries it is likely that the female is the heterozygous sex (like ZW in birds). No instance of XO, or the absence of a Y chromosome in the male, had yet been discovered. It is probable that chromosome determination of sex is widespread in plants, but with no visible distinctions between X and Y in most species.

If sex is dependent on chromosomes in plants, it would be expected that sex-linked inheritance would occur. This expectation has been fulfilled in Melandrium, in which the genes for narrow and broad leaves have been shown to be in the X chromosome.

Sex Genes.—Since chromosomes house the genes for inherited characters in general, the question promptly arose whether the X and Z chromosomes as a whole are responsible for sex, or whether one or more genes for sex are located in them. The most fruitful source of information on which an answer to this question could be based has been the production of translocations and deficiencies (page 205) in Drosophila. Such breakages can be induced by X rays, hence abundant material for study is available. By using flies in whose X chromosomes there are mutant genes scattered along the map, to serve as markers, the location and extent of the aberrations can be roughly ascertained

by breeding. The effect of aberrations at different places on the sex characters of the flies was at the same time observed. From such studies Patterson (1930) had concluded that the sex gene or genes, if any, must lie between the genes for singed (curled irregular bristles, locus 21) and forked (crooked, divided bristles, locus 56.7). This segment constitutes about half of the X chromosome. Dobzhansky and Schultz (1934), however, find that the sex genes must be distributed along the whole X except the inert region near its "right" end.

Genic Balance and Sex.—The conclusion that many genes are concerned with sex is confirmed and extended to the autosomes by the work of Bridges on intersexes and "supersexes" in Drosophila. These modifications of the typical sexes are characterized by various degrees of development, not only of the ovaries or testes, but of the other distinguishing marks of sex—the external genitalia, black bands on the abdomen, sex combs on the front tarsi of the males. Through nondisjunction, flies were obtained that had irregular numbers of chromosomes of the several pairs, and sex was found to be dependent on the ratio of the X chromosomes to the autosomes. A typical female has two X chromosomes and two sets of autosomes; she may be formulated as being 2X:2A. A male, by the same characterization, is 1X:2A. Among the flies with unusual chromosome numbers, those in which the ratio of X to A was 1:1 (as in 3X:3A, 4X:4A, 1X:1A) were all females. When the ratio of X to A was between 1:1 and 1:2 (as in 2X:3A), the fly was an intersex (intermediate between the sexes). If the ratio was more than 1:1 (3X:2A) an exaggerated female (superfemale) was produced, while a ratio less than 1:2 (1X:3A) yielded a supermale.

While the autosomes tended to behave as a group in nondisjunction, the small spherical fourth chromosome sometimes broke away. When this happened, it was found that chromosome 4 assisted the X chromosome in favoring femaleness, while chromosomes 2 and 3 favored maleness. It was a balance of these two groups against each other that determined sex. It seems reasonable to suspect, then, that sex may be dependent on genes in many chromosomes in animals in general.

Also dependent on a balance, but not wholly a genic balance, is the sex of the gypsy moth, as conceived by Goldschmidt. He regards the male-determining genes as residing in the Z chromo-

somes, while the female-determining influence is in the cytoplasm of the egg. A balance between these two determines the sex. Two Z's are enough to override the cytoplasm, and a male is produced. A single Z, however, is dominated by the cytoplasm, and a female results.

Gynandromorphs.—Sometimes, through irregularities of cell division, maturation, or fertilization, animals are produced which have one chromosome combination in one or several parts of the body and another combination in other parts. When

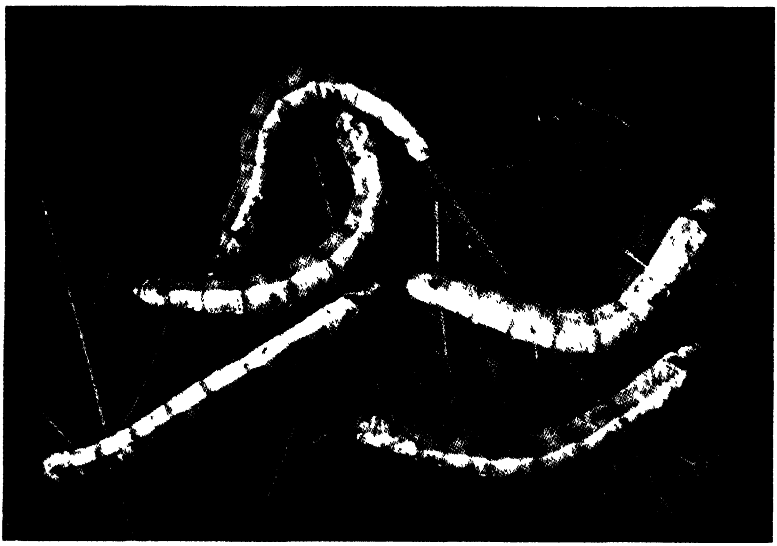


FIG. 106.—Bilateral gynandromorph of silkworm moth larva. (*From Goldschmidt and Katsuki in Biologisches Zentralblatt. Verlag Georg Thieme, Leipzig.*)

these chromosome combinations are sex-determining ones, patches of genetically female tissue may be interspersed among areas which are genetically male. In insects these adjoining areas are able to develop in accordance with their contained genes, and a sex patchwork results. Such mosaic individuals are called *gynandromorphs*. An example is shown in Fig. 106, caterpillars of the silkworm moth which are male on one side, female on the other.

Several possible means of producing gynandromorphs exist. One is through nondisjunction of the X chromosome in the first cleavage of the egg. A gynandromorph of Drosophila was

proved, by mutant genes in the X chromosomes, to be of this kind. At the first cleavage, one of the X chromosomes was lost (did not enter either daughter cell, Fig. 107), so that only one X remained on the left side, while there were two X's on the right. The resulting fly was male on the left side, female on the right.

Another way of producing gynandromorphs is by means of eggs with two nuclei. Such eggs have been discovered in cytological studies. The two nuclei were supposedly present before maturation began, and each is conceived to have gone through the maturation process independently of the other. If this binucleate condition occurs in moths, where the constitution of the female is ZW, one of these nuclei may eliminate its Z chromosome into the polar body, while the other eliminates its W chromosome (Fig. 108). The haploid nuclei are therefore one W and the other Z. If they are both fertilized (necessarily with a Z spermatozoon), one should develop female tissue, the other male. What sort of gynandromorph would result would depend on where the cleavage products of these two nuclei were distributed. The moth larvae in Fig. 106 could have come from binucleate eggs if one combination nucleus went to the left side, the other to the right. Goldschmidt and Katsuki (1927) and Cockayne (1935) are of the opinion that most gynandromorphs in butterflies and moths develop from binucleate eggs.

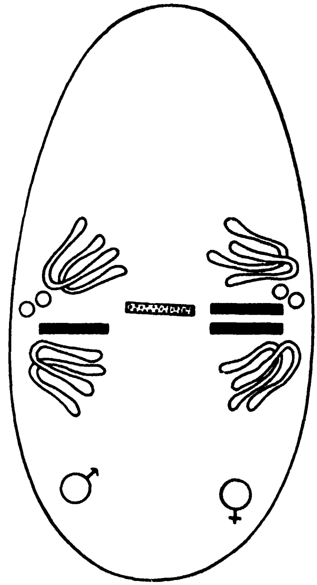


FIG. 107.—Production of a gynandromorph by nondisjunction of one X chromosome in first cleavage of Drosophila. Left side becomes male, right female. X chromosomes are black, except the lost one, which is dotted. (*After Stern.*)

Sex in the Honeybee.—In the honeybee, the eggs may either be fertilized, or they may develop parthenogenetically. The fertilized eggs produce females (queens or workers, depending on how they are fed in the larval stages), while unfertilized eggs yield males (drones). The males are thus haploid, the females diploid.

How sex is related to chromosomes in the honeybee is not exactly known. The female (queen) has 32 chromosomes, and after reduction the mature eggs have 16. The male has only 16 chromosomes and, through an unusual type of maturation which involves cells of very unequal size, and in which there is in effect no reduction, produces spermatozoa likewise with 16 chromosomes. Fertilization of the egg restores the number 32 for the females, while males, developing parthenogenetically, have only 16. While there is a large chromosome difference between the sexes and while the female may very well have two X chromosomes and the male only one, it is difficult to see any different ratio of X chromosomes to autosomes. Haploid Drosophila is still female, because the ratio of X to A is 1:1. Either this ratio does not govern sex in the honeybee, or there is some feature of maturation which escapes detection.

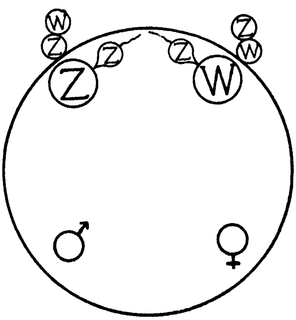


FIG. 108. Maturation of a binucleate egg of a ZW species. Small cells at sides above are polar bodies. The Z chromosome remains in one egg nucleus, the W in the other, after reduction. A gynandromorph, male on the left, female on the right, could develop from it.

Multiple Embryos and Sex.—As a rule one egg, fertilized or parthenogenetic, gives rise to one individual. In some animals, either as a regular occurrence or as an occasional event, a single egg may produce two, a dozen, or hundreds of offspring. The larger numbers occur in the insects; in the armadillo it is four; while in man the number may vary from two to five. In man they are called identical twins (Fig. 109), identical triplets, etc., referring to the fact that they have precisely the same genotypes. The litters of most mammals are not identical, but many eggs are liberated from the ovaries at the same time, and each fetus develops from a separate egg.

The feature of multiple embryos that is of interest in sex determination is that those of one group are all of the same sex. The meaning of this is merely that sex is determined before the embryos start their individual development. Were it not determined so early, the offspring ought frequently to be of different sexes, just as the members of ordinary litters usually include both

sexes. Now, this separation, or at least separate development, of the multiple embryos occurs rather early. In insects, it is already taking place or has taken place when the embryonic mass is in the mulberry stage consisting of some hundreds of cells. In the armadillo, the number of cells in the blastocyst is probably greater than this when the buds that produce the separate individuals arise, but it is still an early stage. In

FIG. 109.—Identical twins so much alike that even their mother often confused them. From their origin from a single egg their genes must be alike. (*From Wiggam in Journal of Heredity.*)

species in which it is already known that sex is determined at the fertilization of the egg, multiple embryos give no new information; they merely confirm the conclusion drawn from chromosomes. When the chromosomes of a species are not known, the sameness of the sex of the multiple embryos gives independent evidence of very early determination.

In insects there are sometimes, in species which produce multiple embryos, broods which include both sexes. These proved very puzzling, and led to various explanations. The evidence now available appears to indicate that two eggs gave rise to the mixed broods. These insects are mostly parasitic

on other insects, and it has been observed that the parasite often lays two eggs in the host. If one has the chromosome constitution of a male, the other that of a female, the resulting offspring should be of both kinds.

Normally Changing Sex.—In the mollusks (snails, clams, oysters) it is common for each individual to be potentially of either sex and to change from one sex to the other as a normal part of the life cycle. The species that have been studied most begin regularly as males, though in rare individuals the

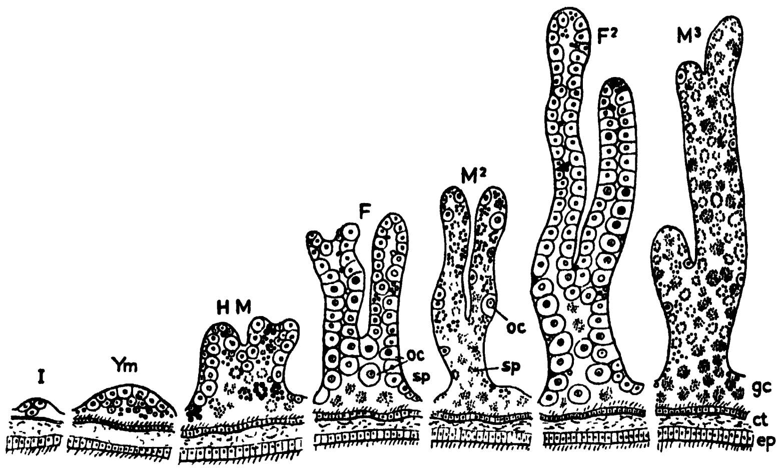


Fig. 110.—Diagram of the sequence of the sexual changes in the gonads of an oyster. Large rounded cells (*oc*), oöcytes; clusters of small dots (*sp*), male cells. *I*, indifferent gonad; *Ym*, bisexual gonad of young animal; *HM*, first male phase, somewhat hermaphroditic; *F*, first female phase; M^2, F^2, M^3, successive changes to male, female, male. *ct*, connective tissue; *ep*, epithelium; *gc*, genital canal. (*From Coe, Mémoires du Musée Royal d'Histoire Naturelle de Belgique.*)

early male phase may be aborted. Later this young male changes to a female; and if it lives long enough, it may be converted into a male again, and finally into a female once more. The changes in the reproductive organ itself, as they occur in a species of oyster, are shown in the successive diagrams of Fig. 110, where the larger rounded cells are oöcytes, the clusters of dots the male cells.

This normal or usual cycle can be modified by external conditions. The common oyster living as far south as Virginia may omit the first male phase and function as a female in its first breeding season (Coe 1936 *b*). Possibly nutrition is the reason

for this haste in passing on to the first female phase. In the shipworm Teredo (a mollusk, not a worm) the functional phase at any time is partly determined, according to Coe (1936 *a*), by age, size, and season of hatching as well as genetic factors; there is, consequently, much individual variation in the course of the sexual cycle.

In Crepidula, a marine snail, the animals influence one another. The young animal is ordinarily male, and moves about freely. Later, while still a male, it becomes more sedentary, finally resting for long periods in one spot. In the species *Crepidula fornicata* there is a strong tendency for the animals to pile upon one another (Fig. 111). The bottom of the pile is commonly an empty "dead" shell, upon which, in successive stories, the living snails are attached. Now, this association of individuals accentuates the early male phase; it is necessary if the male phase is to last more than a short time. If an underlying male is removed from his shell without disturbing the one above, the latter quickly transforms into a female. Usually it is only the one immediately above him that changes quickly; the ones still higher in the pile linger on in the male condition. The reason for this result is that some substance or other influence emanates from the lower individual to the one above. That substance or influence favors continuation of the male phase; removal of it permits transformation to the female phase. Lack of this substance does not cause, but merely hastens, the change, for each individual would become a female eventually anyway. Forced activity is said also to hasten the transformation to a female (Coe 1937).

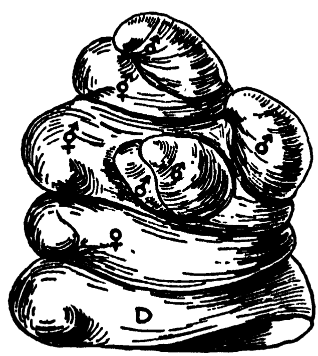


FIG. 111.—*Crepidula fornicata*, individuals piled upon one another, with a dead shell as a base. Sexes are indicated; the combination sign means a snail in sex transition. (*From Coe in Journal of Experimental Zoology*.)

Sex Influenced by Environment.—The influence exerted by environment upon sex in mollusks serves rather to alter the speed of a change which would occur anyway, than to bring about an event that would not otherwise happen. Somewhat the same kind of influence, in that the tempo of events is changed, is

exerted by environment in the worm Bonellia but here the final results may be altered. Bonellia exhibits an extreme sexual dimorphism. The female has a rounded body, with a long proboscis divided at the end, the whole animal being several inches in length (Fig. 112, *A*). The male is a very degenerate

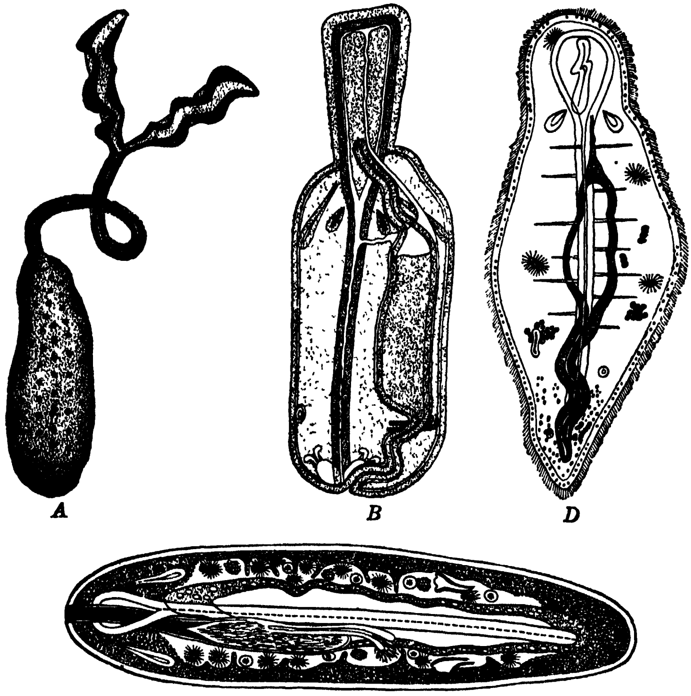


FIG. 112.—The worm Bonellia. *A*, adult female; *B*, young female; *C*, male; *D*, intermediate form, resembling the male chiefly, but having some female characteristics. The young larvae have the capacity to become either sex. *A* is somewhat reduced, all others greatly magnified. (*A original; B-D modified from Baltzer.*)

animal of minute size (*C*). The young larval stages have the capacity of developing into either sex. If they become attached to the proboscis of an adult female, on which the males live parasitically, they become males. Those which fail to reach a proboscis develop more slowly and usually become females (*B*). Some substance must pass out from the proboscis of the female to govern the change. Larvae which become attached so late

that development in the female direction has already started often become intermediate with respect to sex (*D*).

Another group of animals in which environment controls sex, though rather indirectly, is the rotifers. These minute aquatic forms have two kinds of females, looking exactly alike but differing in their modes of reproduction. One kind produces eggs which develop parthenogenetically and always yield females. The other kind produces eggs which can be fertilized but which can also develop parthenogenetically. If these latter eggs are

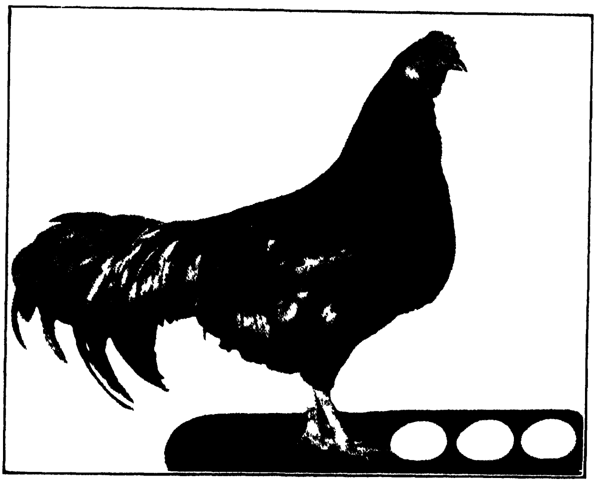


FIG. 113.—A hen with cock-like tail feathers developed presumably as a result of disease in the ovary. The ovary resumed its normal functions and eggs were produced, and at the next molt short tail feathers were produced (see Fig 114). (*From Cole in Journal of Heredity.*)

fertilized, they yield females; if unfertilized, males come from them. The only way to get males is first to obtain the kind of females whose eggs can be fertilized and then to prevent the eggs from being fertilized. The first step, production of the male-producing females, can be largely controlled by environment. Many chemical substances in the water in which the rotifers live help to prevent the production of these females, hence the number of males is reduced; on the other hand oxygen in the water and certain changes of food increase the frequency of this type of female and consequently the number of males.

Secondary Sexual Characters.—The organs that carry on the actual functions of reproduction are known as primary sex

organs. Those that distinguish the sexes from each other but play no *direct* part in reproduction are called secondary sexual characters. There is usually a close connection between the two, and the phenotype which is ordinarily designated male or female is a combination of primary and secondary sex characters. Examples of male secondary characters are the long tail feathers and spurs of fowls, the mane of the lion, the brilliant plumage of many birds, and the beard and baritone voice in man.

In vertebrate animals the secondary sexual characters are mostly under the control of hormones produced by the testes or

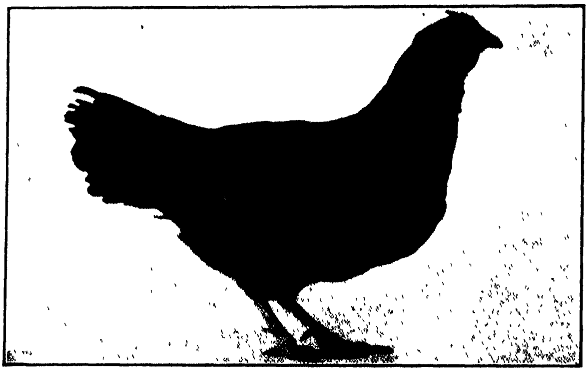


FIG. 114.—The same hen as in Fig. 113, but nine months later, after the fall molt. Short tail feathers have replaced the long ones, indicating a restoration of normal function of the ovary. (*From Cole in Journal of Heredity.*)

ovaries. These hormones appear to be produced, not by the germ cells themselves, but by the interstitial cells which surround them; for treatment of the organs with X rays of proper dosage may inactivate the germ cells, at least with respect to their reproductive function, without interfering with the production of the hormone. Absence of the proper hormone at an early stage may prevent the development of the expected secondary characters. Thus, castrated male Brown Leghorn fowls do not develop their combs and wattles beyond the juvenile stage. Many other examples of such interference with normal development are on record.

Sometimes the interference arises without man's intervention. Cole describes a fowl which must originally have been a hen, but which when first observed had long tail feathers like those of a cock (Fig. 113). While in this partially male condition the fowl laid eggs from which normal chicks developed. After the

next molt, however, the tail feathers were short (Fig. 114), and the fowl was a typical hen. The probable explanation is that some disease, perhaps tuberculosis, practically destroyed the ovary so that the usual female hormone was lacking. When, in this condition, new feathers developed after a molt, long tail feathers were part of the equipment. Then the ovary recovered, and eggs were laid as described, but the long tail feathers necessarily persisted until the next molt.

An important difference between secondary sex characters and ordinary genetic characters is shown by some transplantation

FIG. 115.—Patch of skin from a barred male fowl grafted upon a self-colored female, before the feather characters had developed. (*From Danforth in Journal of Heredity.*)

experiments of Danforth. He removed a patch of skin from the back of a young female fowl destined to be of solid color, and grafted in its place skin of a barred male. When the adult feathers developed, there was a patch of barred plumage in the midst of self-colored (Fig. 115). The color pattern is an ordinary genetic character. The secondary sex character involved is the shape of the feathers. In the male the feathers taper off to a point, while in the female the ends of the feathers are rounded (Fig. 116). When the transplanted patch was examined, it was found that the feathers were rounded. Though these feathers came from a male and would there have been pointed, they assumed the rounded form on a female. Mere

transplantation was not the cause of the change of shape, for skin grafted from male to male bore pointed feathers (Fig. 116, *B*). Pattern is genetic in the ordinary sense; shape of feather is a secondary character. Strictly speaking, the secondary characters are likewise genetic; but since hormones are part of the mechanism of their development, it is relatively easy to bring about environmental modification.

A *B* *C* *D*

FIG. 116.—The difference between ordinary inherited characters and secondary sexual characters. *A*, feather produced by skin of a black female transplanted to a barred male; *B*, from a barred male transplated to another barred male; *C*, feather from the female fowl shown in Fig. 115, developed from her own skin; *D*, from the transplanted patch of skin on the fowl of Fig. 115. (*From Danforth in Journal of Heredity.*)

Sex Reversal.—Occasionally environmental influence may completely reverse the sex of an individual, both as to gonads and ducts and as to secondary characters. A remarkable instance of reversal is that of an adult hen which became a cock, as reported by Crew. This hen had laid normal eggs from which chicks hatched. Then she began to develop the secondary characters and the behavior of a male (Fig. 117). When the change appeared completed, the fowl was used as a male, and actually fathered normal chicks. The reversed fowl was a Buff Orpington, and, to prove that it was actually the father of the chicks produced, it was mated to a hen of another breed. The chicks had Buff Orpington characters, which their mother,

even if she had previously mated (which could have happened only with males of her own breed), could not have given them. Postmortem examination of the reversed fowl showed that the original ovary had been destroyed by a tumor caused by tuberculosis, and for some reason a testis had been produced by regeneration in its place.

Sex reversal has been produced in many animals—amphibia, chicks, fishes—to the extent of changing the histological character of the gonads and to some extent the ducts, but the change has seldom gone far enough, and in an adult animal, to permit the

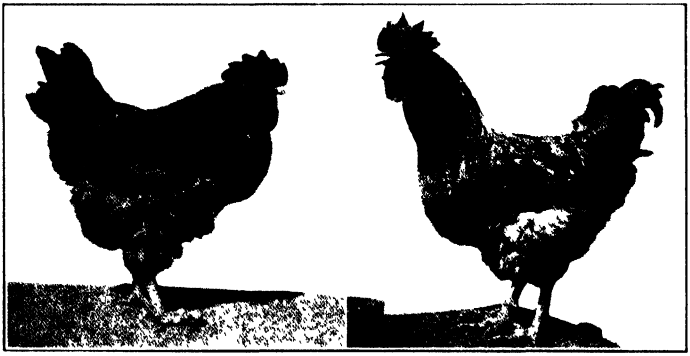


FIG. 117.—A Buff Orpington hen that became a cock. Two stages, early and late, in the transformation of a female into a male. This hen had laid eggs which produced normal chicks before this transformation took place. About four months after the second picture was taken "she" became the father of two normal Buff Orpington chicks, one male and one female. (*From Crew in Journal of Heredity.*)

individual to function as of the new sex. Crew's example and one or two others are the only ones so complete.

Plants which are of one sex only (not hermaphroditic) can also be changed to the opposite sex in some species. Hemp, for example, has been converted from male to female or female to male by altering the length of day.

Intersexes.—Incomplete sex reversal often leads to the production of individuals intermediate between the sexes. Usually such organisms are patchworks in a sense, because their various parts are not intermediate to the same degree. Some structures may be more like the female, others more like the male. Individuals like this are not gynandromorphs, because the parts are not definitely and clearly male or female; the parts, or at least

some of them, are intermediate. These intermediates are known as *intersexes*.

The nature of intersexes has afforded a clue concerning the manner of their production. Goldschmidt, in intensive studies of the gypsy moth, has offered the following explanation. An intersex is an individual which started development as of one sex, experienced a change of developmental physiology at some point, and thereafter proceeded to develop as of the other sex. The various structures which distinguish the sexes have their fate decided at different times. If an embryo starts developing as a male, any sex organs having their future decided early will be of the male type. Then the physiological change takes place, development is thereafter that of a female, and any organs determined later will be female. Since either the physiological change is gradual, not sharp, or the determination of the fate of structures is spread over an appreciable time (not momentary), some or all of the structures resulting are intermediate. An intersex gypsy moth is shown in Fig. 118 between the typical sexes. The intermediacy is most apparent in the pattern of the wings but is observable also in the size of the body and in the antennae.

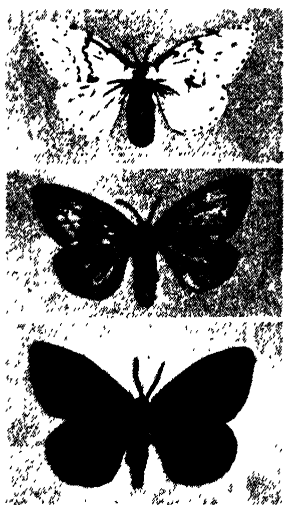


Fig. 118.—Gypsy moth intersex, between typical female (above) and typical male (below). (*From Goldschmidt in Ergebnisse der Biologie.*)

What are essentially intersexes are produced in cattle through hormone action in fetal development. When twins are produced, they may be both males, both females, or one of each sex. In any of these cases, the embryonic blood vessels may be separate, or may join to a greater or less degree. If one twin is male, the other female, and if their blood vessels anastomose anywhere, the blood of the male may flow through the vessels of the female, or vice versa. The male fetus develops a little faster than the female, and arrives earlier at the stage in which the sex hormones are produced. The male hormone thus travels in the vessels of the female before any female hormone is present. The effect of

the male hormone is to arrest female development, and the female fetus never does reach the stage of hormone production. The original female thus continues its development more or less as of the male type and becomes an intersex. An intersex which is made so by action of a hormone of the opposite sex is called a *freemartin*. In cattle the freemartins are all modified females.

In man there are anomalies of sex development, traceable to deficient or at least abnormal hormone action. Both internal and external sex organs and secondary characteristics are modified. Such abnormal individuals have often been called hermaphrodites, but intersex is the right name.

Intersexes, whether in man or other animals, are almost invariably sterile.

Sex Ratio.—The chromosome mechanism of sex determination, wherever it consists of X and Y or Z and W chromosomes, should lead to a ratio of males to females of about 1:1. When, as in the honeybee, sex depends on whether the egg has been fertilized or not, the sex ratio is determined by the number of eggs fertilized. The queen bee can control that ratio, following some sort of stimulus, and the females are regularly in a very great majority. When sex is dependent on the environment, the ratio is apt to fluctuate enormously.

Even in organisms with X and Y chromosomes the sex ratio may fluctuate. It may even be regularly one-sided. In man, for example, there is in almost any population of considerable size a small excess of males. Such deviation is not necessarily, however, a matter of sex determination. It may be only differential production or survival. Experiments which shifted the sex ratio, at first thought to indicate that sex determination had been controlled, turned out later to show only that one sex had survived the experimental conditions better than the other.

It was once proposed that the surplus of males in the human race was due to this selective survival. When the only available evidence on this point was obtained, namely, that from stillbirths, it appeared, however, that the females were not more subject to such early mortality. The difference is indeed in the opposite direction. Some statistics collected by Greulich (1931) relating to over 50,000 stillbirths in a selected area of the United States showed the following distribution of sex:

Age of Fetus, Months	Relative Number of ♂♂ ♀♀
Less than 4	357 48:100
4	223 14:100
5	139 36:100
6	128 91:100
7	116 60:100
8	125 26:100
9	137 43:100
10 or over	150 13:100

Unless there are unrecovered losses which are preponderantly females, the excess of males at the time of fertilization is greater even than in the living population. Presumably more eggs are fertilized by the Y-chromosome than by the X-chromosome type of spermatozoon. It has been claimed that treating the spermatozoa with certain chemical substances disabled one of these classes of male cells more than the other. If something exists naturally, perhaps in the eggs, that would favor one kind over the other, the sex ratio could be made one-sided. Such an influence could hardly be called sex determination, however.

CHAPTER XXII

HEREDITY AND EVOLUTION

Despite their conservatism, vouchsafed them by their genetic mechanism, the types of organisms on the earth have undergone considerable change through long periods of time. There are many indications that the hundreds of thousands of species now in existence have sprung from relatively few origins of life, perhaps even from only one origin. These indications come from similarities of structure, of development or of physiology, from distribution on the earth, and from fossils. With practical unanimity, the evidence points to a very extensive evolution of living things. How has this change come about?

From all that can be seen happening now, or inferred from the end results of past occurrences, evolution is and has been slow. When any change is effected, it lasts for a long time as a rule. The scheme of living things is plastic but is also resistant. There is nothing chaotic about evolution; it is certain there are brakes upon it. The reasons for both the existence of evolution and its slowness are matters of some importance.

Heredity may be counted upon to furnish the resistance. The protein nature of the genes and the very specific nature of their actions provide a strong tendency to maintain the *status quo.* If, then, change is possible, and if alterations may themselves become semipermanent through heredity, the evolution process is largely explained. Heredity and evolution used to be regarded as antithetic phenomena. One was thought of as undoing the work of the other. Difficulty was experienced in conceiving of heredity taking up the changes produced in evolution and making them permanent.

All this difficulty was gradually removed with development of understanding of the Mendelian mechanism. It was realized that the changes which constitute evolution occur in the genetic mechanism itself. The mechanism of heredity is also the mechanism of evolution. It is responsible for both the modifications

and their permanence. The problem of evolution is first of all to discover how the genetic mechanism operates to bring it about.

Primary Sources of Variation.—The primary method of producing change is through *mutation,* or modification of individual genes. Other sources of variation exist, but they mostly depend on pre-existent mutation. The genes are chemical substances of a highly complex composition, hence are subject to alteration. Part of this alteration is presumably transitory, being part of the physiological processes, and means nothing in evolution. Some changes, however, are permanent or semipermanent. When the gene at locus 104.5 in chromosome 2 in Drosophila

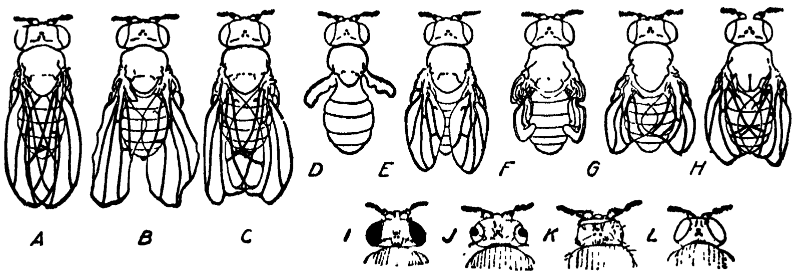


FIG. 119.—Some of the modifications which have arisen in the vinegar fly *Drosophila melanogaster* since 1910. A thousand others have been observed. *A*, normal wing; *B*, beaded wing; *C*, notch wing; *D*, vestigial wing; *E*, miniature wing; *F*, club wing; *G*, rudimentary wing; *H*, truncate wing; *I*, normal red eye; *J*, bar eye; *K*, eyeless; *L*, white eye. (*C from Morgan; D and L original; the rest adapted from Morgan, Sturtevant, Muller and Bridges, Mechanism of Mendelian Heredity, Henry Holt and Co. From Shull, LaRue and Ruthven, Principles of Animal Biology, McGraw-Hill Book Company, Inc.*)

changes in a particular way, a fly that has two such modified genes develops brown eyes instead of red. The wild-type gene has mutated. That change took place many years ago, and the new gene has remained unaltered through a long line of descent ever since.

Hundreds of genes have mutated in Drosophila since the year 1910. Probably they had been doing so earlier. The structures modified are the eyes, wings, body, bristles, and legs. A few of the mutated characters are shown in Fig. 119. Corn also has experienced such changes in every part of the plant. Smaller numbers of mutations have been observed in mice, rats, guinea pigs, and rabbits. Every animal or plant that is raised in large numbers under careful scrutiny has produced mutations. It is safe to assume, therefore, that modification of genes is so wide-

spread that evolution may be regarded as starting in that manner.

Other methods of introducing change are either minor or secondary. One of them is change of position of genes. While genes have a definite arrangement in the chromosomes which is regarded as normal, their relations to one another may be disturbed by translocations, inversions, duplications (page 115), and unequal crossing over. The first "position effect" was found by Sturtevant (1925) who discovered that when the Bar-eye segment (page 116) of the X chromosome was involved in unequal crossing over, so that both of them were in one chromosome and none in the other, the reduction of the size of the eye was greater than when each chromosome had one of the segments. The same genes were present in the fly in either case but were more effective if concentrated in one chromosome than when they were in two. A number of other position effects have been found since then. A gene in one position in the chromosomes does not always do quite the same work as in another position; the characters of an animal or plant may thus be changed merely by rearranging the genes.

A rather important source of change is the doubling of the chromosome number in the cells. Any change in the number of chromosomes is apt to modify the nature of the individual in which it occurs. Even a change of only one in that number is often noticeable in physical appearance. We have already observed how the loss of one fourth chromosome changes the appearance of Drosophila (page 114). In the Jimson weed Datura, 12 different types of plants result from the addition of only one chromosome in each. There are 12 pairs of chromosomes in Datura, and the addition of an extra 1, making 3 of that kind, changes the plant in a different way for each of the 12 chromosomes. The modification is seen in the shape of the spiny seed capsule and other features. Addition or subtraction of 2, 3, or 4 chromosomes has comparable effects. The reason why doubling the chromosome number is singled out for special mention is that through it a new viable type may become established very quickly. Because of the balance among the chromosomes which is maintained when the number is doubled, such forms are more successful than are those resulting from irregular chromosome changes. One of the very common

effects of this doubling is larger size and more vigorous growth, as observed in evening primroses, Jimson weeds, tomatoes, and other plants. New races and species have originated at a single step by this method. Varieties of Petunias have sprung from other varieties by doubling the chromosomes; the tape grass or wild celery, *Vallisneria spiralis*, with its 20 chromosomes produced another species *V. gigantea* with 40 chromosomes; *Oenothera lamarckiana* with 14 chromosomes produced *O. gigas* with 28 chromosomes, etc.

Recombination.—Once a multiplicity of unlike genes at various loci have arisen by mutation, the chief source of further variation is recombination. The genes are brought together in ever new combinations as rapidly as crosses are effected between individuals differing with respect to them. Mutation does not stop, but a greater amount of difference among individuals is derived from recombination than from changes of genes.

With the general principles of recombination we are already familiar. It takes place whenever two animals or plants differing in two or more characters are mated. It is freer if the characters are independent of one another (genes in different chromosome pairs), but linkage is no barrier to the ultimate production of every possible genotype. All of the combinations expected from a certain mating may be realized in somewhat fewer generations if the genes are independent than if they are linked, but in a long-range phenomenon like evolution a few generations are unimportant. Allowing even the greatest possible hindrance by linkage, variation by recombination should still occur more rapidly than the new types can fit themselves into the environment.

Were mutation to cease, recombination would finally reach a qualitative conclusion, in that all possible genotypes would exist. A population in which this had happened would eventually include a certain proportion of each of the genotypes. What proportion of genotype there would be would depend on the relative abundance of the alternative genes. Thus, if in a population, 90 per cent of the chromosomes of pair 1 had gene *A* and 10 per cent gene *a*; if 75 per cent of the chromosomes of pair 3 had gene *B* and 25 per cent gene *b*; and if 60 per cent of chromosomes of pair 7 had gene *M* and 40 per cent gene *m*; then each of the 8 phenotypes, or the 27 genotypes, into which the genes

Aa, *Bb*, and *Mm* could enter should exist in a certain calculable fraction of the population. If, however, the respective percentages were 80 and 20, 45 and 55, 17 and 83, the fraction of the population represented by each genotype or phenotype would be different. An equilibrium would be established, and the proportions of the different kinds of individuals would remain the same through successive generations. All this is based on the assumption that the various kinds mate at random, that all are equally fertile, and that none of them possesses any advantage over the others.

Such an equilibrium never actually exists. It is only an ideal condition from which to measure fluctuations or more permanent changes. The equilibrium is repeatedly upset by new mutations, which permit more combinations to be formed. Some of the genotypes turn out to be more frequent than is expected, purely as a result of chance. Moreover, mating is probably never random, for in most species, owing to restricted travel, each individual is limited in its choice of mates to those in its neighborhood. Under these several conditions, if something happens in one area of a species—a new mutation, accidental excess of one genotype—there is bound to result, in that immediate neighborhood, a redistribution of the proportions of the various genotypes. Something like a local race arises, even though there is no visible change in the organisms.

With all these factors working against an equilibrium, recombination of genes goes on continuously. As stated earlier, it is the principal source of change after mutations have accumulated.

Hybridization of Species.—The recombination just described results from hybridization among individuals having to some extent different genes. A somewhat more radical recombination is effected when organisms belonging to different species can be and are crossed. Although individuals of the same species may differ in one or several genes, those of different species may be unlike in dozens of them. It is not always, not even usually, possible to cross species, for there is a strong tendency for species to be intersterile. Some of them will not mate, or their germ cells will not unite, or the hybrid does not reach maturity. Other species leap all these hurdles, cross, and yield offspring, but the hybrids are sterile. There are, however, many grades of interfertility between species, and some such

crosses are as fruitful as matings within species. When there is high fertility, recombination of the characters of the two species occurs about as freely as recombination within a species.

One difficulty in species crosses arises from differences in the chromosomes of the two species. These chromosomes must be paired in the hybrid in order that normal germ cells may be produced, and similarity of the genes in them is conducive to pairing. In a hybrid whose two chromosome complements are unlike, there are many irregularities in the distribution of the chromosomes in maturation, and many germ cells which are deficient in chromosomes or have extra chromosomes. These cells do not function normally, so that dissimilarity of chromosomes from the parents is one of the causes of partial sterility of hybrids.

This difficulty in the pairing of the chromosomes has been solved in a number of species hybrids by the expedient of duplicating the chromosomes. Even when the crossed species have different numbers of chromosomes, the duplication of them provides like pairs. Thus, if one species has 18 chromosomes (the haploid number $n = 9$) and another has 24 ($n = 12$), the hybrid would have 21, but they could not be matched in pairs. However, if these 21 chromosomes are duplicated, there are at once 21 pairs of identical chromosomes, and pairing before maturation should be facilitated. It has been suggested (Clausen 1928) that ordinary tobacco, *Nicotiana tabacum*, originated from a cross between *N. sylvestris* and *N. tomentosa*, or between *N. sylvestris* and *N. rusbyi* (Brieger 1930) in which the chromosomes of the hybrid were duplicated.

Cause of Mutation.—While mutations are presumably the material with which species are created, there is little information concerning the reason for such changes under natural conditions. That the alterations must be chemical modifications has been assumed. Production of mutations by artificial means is not at all difficult. The first highly successful attempts of this sort were those of Muller who, by X-ray treatment, speeded up the production of lethal mutations in Drosophila to 150 times its normal rate. Structural mutations were subsequently induced by the same treatment, such as the character "scute" in Drosophila (Fig. 120). Other types of radiation have likewise proved effective. Radium has been so used in barley, Datura,

the evening primrose (Brittingham 1936), snapdragons, and others; ultraviolet has produced mutations in Drosophila, snapdragons, and corn (Stadler and Sprague 1936); and high temperatures applied to the larvae induced such changes in Drosophila. Age of pollen or seed determines the number of mutations in some plants. The seeds of snapdragons yield several times as many mutations at an age of 7 to 9 years as they do if germinated promptly (Stubbe 1935), and Datura seeds many times as many at an age of 7 to 10 years (Avery and Blakeslee 1936). Mutations causing pollen abortion occur much more often in old pollen of Datura than in fresh pollen (Cartledge, Murray, and Blakeslee 1937). And finally, X rays were found more effective in producing mutations in Drosophila if the flies

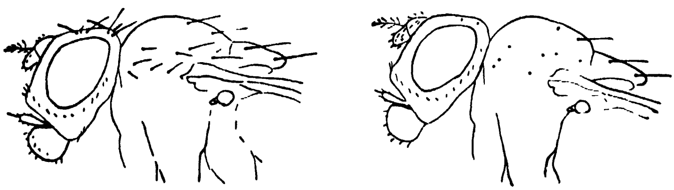


FIG. 120.—Visible mutation produced by X rays in *Drosophila melanogaster*. Normal fly at left, "scute" mutation (absence of certain long spines on head and thorax) at right. (*From Serebrovsky and Dubinin in Journal of Heredity.*)

were fed on cultures containing lead salts or if lead salts were injected into the flies, than in untreated flies (Medvedev 1935).

At first it seemed possible that radiation might be responsible for mutations occurring naturally, since the earth sends forth such rays at all times. Some calculations based on the intensity of this radiation showed, however, that there is not enough of it.

Other environmental agents may produce changes in germ cells, but these are not always so obviously mutations. If guinea pigs are made to inhale alcohol fumes repeatedly over long periods, they produce an excessive number of degenerate offspring—stunted (Fig. 121), blind, deformed, or paralyzed animals. The degeneracy is handed on to a percentage of the later offspring, but the defects gradually die out. Whether these changes occur primarily in genes is not certain.

Because of its historical interest, mention should be made of the old view that environment in general might cause mutations by modifying the body in some respect and that this altered body would then produce mutations in the germ cells within it.

This idea has been generally abandoned, but an example showing how it might have arisen will be useful. In the sea-squirt Ciona water travels through the body, entering and leaving by two projecting tubes called siphons. Kammerer cut off these siphons in a number of individuals, and allowed them to regenerate. The regenerated siphons were longer than those removed. He then obtained offspring from the regenerated animals and found that these likewise had long siphons. Kammerer supposed that the long siphons of the offspring were inherited as an

FIG. 121.—Effect of alcohol on guinea pigs. These two animals were born in the same litter, the mother being descended from alcoholized grandparents. The one at the right was stunted and paralyzed and lived only three days. (*From Stockard in Interstate Medical Journal.*)

acquired character, which would mean that the regenerated siphons of the parents had induced a long-siphon mutation in one or more of the germ cells in the bodies of the parents. The experiments were repeated by Fox who was unable to obtain the same results. He discovered, however, that individuals especially well fed grew long siphons (Fig. 122). It seems likely that Kammerer had cared for his regenerating animals and their offspring better than those with which he compared them and was deceived into thinking that the nutritive effect was the result of a mutation induced by an altered body.

The experiments of Jollos in which heat treatment of Drosophila larvae induced mutations several generations after the application of heat ended (page 212) might be regarded as the

induction of mutations by an altered body. This cytoplasmic effect, however, was temporary. Evolution could not proceed far with that sort of mutation.

Direction of Mutation.—The remaining problems of evolution, as related to genetics, are mainly those of guidance. What has caused evolution to take the course it has followed? It has produced the thousands of species existing at the present time, when it might conceivably have led to organisms of very different kinds. The causative agents might have been very much the same as they have been and yet have produced in the end species quite different from any we now have.

FIG. 122.—A sea-squirt, *Ciona intestinalis*. At the left, typical animal. At the right, similar individual after nine days of especially abundant nutrition, showing the elongated siphons. (*From Fox in Journal of Genetics.*)

The first step in the guidance of evolution has been the guidance of mutation. Evolution cannot go in directions in which there are no mutations. It is quite clear that mutations are not happening in every conceivable way and that some mutations which do happen are more abundant than others. That is, the *direction* of mutation is limited by something.

It is to be expected, from the chemical nature of the genes, that they can be modified in some ways, not in others. All chemical substances are similarly restricted; they enter into certain reactions, not into others. Their chemical structure is the reason for this limitation.

Parallel mutations in different species illustrate further the restriction on direction of mutation. If the sorts of mutation that arise are not subject to some guidance, the mutations of one

species should not show any particular resemblance to those of another species. Yet such correspondences are common. In two species of Drosophila, *D. melanogaster* and *D. simulans*, the similarity is very marked. In each of them there have appeared prune, white, ruby, and garnet eye-color mutations; in each of them a yellow body-color mutation; in each of them crossveinless and rudimentary wings; in each of them forked and bobbed bristles; and some others. When their chromosomes are

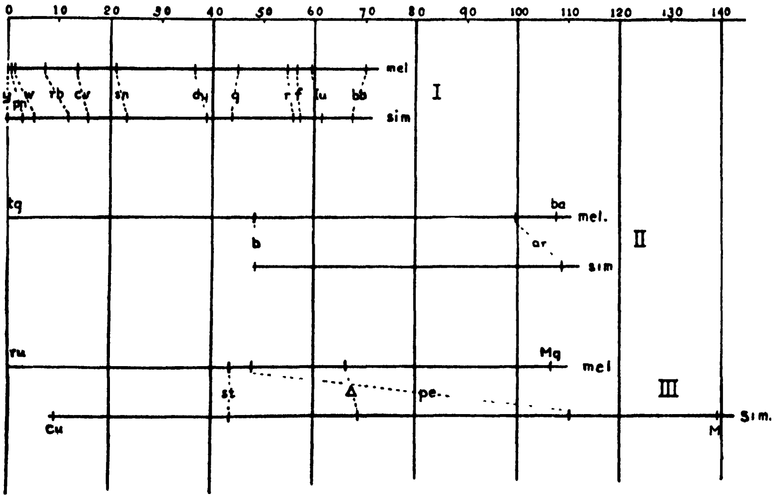


FIG. 123.—Maps of three corresponding chromosomes of *Drosophila melanogaster* and *D. simulans*. Many of the mutations produce the same characters in the two species, and in the main the genes are in the same order in the chromosomes. The correspondence is closest in chromosome I (the X chromosome) in which the mutations to yellow, prune, white, ruby, crossveinless, singed, dusky, garnet, rudimentary, forked, fused, and bobbed are shown. (*After Morgan, Bridges and Sturtevant, in Bibliographia Genetica.*)

mapped (Fig. 123), it is found that these genes are in about the same order in the chromosomes of both species. One might at first suppose that each of these mutations arose just once, in either species, and then was transferred to the other by hybridization. But that cannot happen; for though these species can be crossed, and offspring obtained, these offspring are completely sterile. The mutations that are found in both species must have originated in them independently. That so many of these mutations should be identical is inconceivable, unless the direction (nature) of mutation is being guided. Presumably the chemical structure of the similar genes furnishes this guidance.

Other indications that mutation is directed are the repeated occurrence of the same mutation (scores of times for the white-eye mutation of Drosophila); and reversibility of mutation (the tendency of a mutated gene to go back to its original form rather than to some other new one). Neither of these things should happen if mutation were purely random as to nature (direction).

Chance in the Direction of Evolution.—When mutations have been produced, they begin at once the process of combination with other genes. If they enter into these combinations purely at random, it is expected that each combined genotype will exist in a certain fraction of the population, as explained in an earlier section. The expected fraction is, however, seldom realized. The actual proportion is usually something else, above or below expectation. This fluctuation is due to "chance." The genes may not enter into the germ cells in quite the expected proportions, the germ cells that are fertilized may not be drawn from the various classes in proportion to their numbers, the individuals that mate may not be taken proportionately from the several genotypes.

After the offspring are produced, there is a good deal of accidental elimination among them. Many are lost because they happen to be at a certain place where a destructive event (drying of a pond, a forest fire, for example) occurs—an event over which they have no control and from which they could not escape through any individually different qualities which they might possess. By means of these fortuitous occurrences, a sort of evolution *may* occur. Relatively more individuals of genotype *AaBBCc* may exist in this generation than in the last, and they *may* be even more abundant in the next. Though not highly probable, a succession of changes in the same direction may occur, redounding to the advantage or disadvantage of some particular class.

Changes may happen in one part of the range of a species, not at all or in the opposite direction in another part. When a mutation occurs, it must arise at some one place. Since migration in most organisms is neither rapid nor extensive, the mutated gene and its descendants remain for a long time in a limited area. Furthermore, in the migrations that occur individuals of one genotype may accidentally collect more abundantly in one place than in another. As a result of either of these processes,

a species may become divided into a number of mildly different local races, purely by accident.

Although an extensive evolution can hardly result from chance alone, Wright (1931), after a careful mathematical study of the random operations of the genetic mechanism and the random behavior of animals, is of the opinion that varieties of a species, or perhaps even species of a genus, might arise in this way.

Selection.—The remainder of the guidance of evolution is largely a question of choice among classes of individuals. The genes possessed by each generation must come to it from its parents. If for any reason the parents possess one genotype more frequently than another, the offspring will have more abundantly the genes of that class. Any tendency of one genotype to produce more than its share of the next generation must result in an increase in the frequency of its genes. To illustrate concretely, suppose that in any generation, at a certain locus in the chromosomes of a species, 80 per cent of the chromosomes contain gene A, 20 per cent gene a. Random matings among the individuals, and equal fertility of all of them, would make the following generation consist of the product $(.8\,A + .2\,a)(.8\,A + .2\,a)$, which is $.64\,AA + .32\,Aa + .04\,aa$. The genes are still in the ratio of $8\,A : 2\,a$. With random mating and equal fertility, the ratio remains the same indefinitely.

Suppose, now, that in some generation reproduction is not random. Assume that, among the individuals that reproduce, the heterozygotes (Aa) make up not the expected 32 per cent but 40 per cent, that the AA group drops to 55 per cent, and that the aa group rises to 5 per cent. Only 75 per cent of the genes at this locus in the parents are now A, 25 per cent a, and the next generation should consist of $(.75\,A + .25\,a)(.75\,A + .25\,a) = .5625\,AA + .375\,Aa + .0625\,aa$. The homozygous recessives (aa) have risen from 4 to $6\frac{1}{4}$ per cent, the heterozygotes have increased, but the AA group has declined.

The above changes in proportions of classes of individuals resulted from a disproportionately large number of two classes and a smaller proportion of the third among those which reproduced. Precisely the same sort of change will occur if all individuals reproduce, but some of them are more fertile (produce more offspring) than others. Differences in fertility among individuals of a species are quite common. If increased fertility

is due to certain genes, those genes should increase in frequency from generation to generation. As the favored genes increase, their alleles diminish in number and are finally bred out of the species.

Any gene which causes its possessors to leave more descendants is said to possess a selective advantage. It may do this by increasing fertility, by saving lives that would otherwise be lost, or by merely prolonging life if the reproductive period is likewise prolonged. The only advantage which a gene can confer that is of any significance in evolution is ability to leave more descendants. Ease in getting food, facility in escaping enemies, attractiveness to the opposite sex are all advantages in evolution only if they result in a larger relative number of descendants.

For further discussion of evolution through the operations of the mechanism of heredity reference is made to the work of Wright, cited above; to Fisher (1930) who, however, has made the mistake of assuming that mutations of all conceivable kinds occur; and Haldane (1932).

Isolation.—One peculiarity of species is not accounted for in the foregoing description of the evolution process. That is the rather sharp definition of each species from other species. Although differences within a species merge gradually into one another, those between species are mostly rather sharp. An individual can as a rule be assigned to a certain species without question because of this clear separation. The sharp definition is due to the absence or scarcity of intermediate forms.

On the whole the distinctness of species results from the absence or infrequency of crossing between them. Sometimes the lack of crossing is due to geographic separation of the ranges. Occasionally there is an impassable barrier between them. More frequently, however, the species could intermingle freely and still be isolated genetically. This isolation results from the inability of different species to breed with each other. Although as pointed out earlier there are species that cross freely and some from whose hybrids new species have been known to arise, most species are unable to cross at all or at best produce partially or completely sterile hybrids.

Were it not for this isolation, the distinctions between species would largely disappear; the gaps would be bridged over by intermediate individuals. Whether the merging of species into

one another would be an advantage or a disadvantage is uncertain. Probably it would make little difference one way or the other in the success of the organisms. But one very characteristic feature of living things would be removed, and the problem of the classifier would either be much more difficult or not exist at all.

How isolation (other than geographic) is brought about is known for only a few species. As between *Drosophila melanogaster* and *D. simulans*, whose mutations and chromosomes are so similar, the separation is due to what may be regarded as sterility genes. Sterility arises from the interaction of two dominant genes of different pairs. One species has one of these dominants, the other species the other dominant. Each species is fertile within itself, but in their hybrid the interacting genes are brought together and sterility results. In some other examples, sterility is due to unmatched chromosomes in the hybrid. The chromosomes do not pair properly and are distributed irregularly in maturation. While some germ cells should accidentally receive a viable chromosome combination, many would not, and partial sterility would result. When species mate but do not produce any offspring or when they do not mate, the difficulty may be chemical (within the germ cells), or structural, or psychological. Species whose germ cells mature at different seasons are very effectually isolated, even though living in the same region.

CHAPTER XXIII

INHERITANCE OF HUMAN STRUCTURAL CHARACTERS

A few human characters have already been presented at various points, where they appear to illustrate certain types of genetic phenomena. It is desirable now to describe briefly the salient features of a number of other traits. These are assembled in the next several chapters. For convenience they are classified as structural, physiological, and mental. This division is largely arbitrary, since structure arises out of the physiology of development, physiology differs according to structure, and mental qualities rest on the structure and physiology of the brain. Characters are listed as structural if they are detected chiefly by observation in the absence of any particular activity, but the line between structure and function is not easily drawn in many instances.

Whether and How.—Because of the relatively meager information which single family histories give and because it is never possible to assume that two or more family histories begin with the same genotypes even when the phenotypes of the parents are the same, knowledge of human heredity is never comparable with that of other organisms. All information is valuable, but conclusions must vary in proportion as it is complete or partial. As a result, it is often possible to discover *whether* a character is inherited when it is impossible to learn *how* it is inherited.

When a character occurs repeatedly in some pedigrees and seldom or not at all in certain others, there is a rather plain indication that it has a genetic basis. When there is distinct correlation (see Appendix) of the incidence of a character in parents and offspring, or in brothers or sisters, this too is evidence that the quality is inherited. These two tests have long been used to ascertain whether a trait is genetic, even if its mode of inheritance (whether dominant, recessive, blending, etc.) cannot be determined. In recent decades another means of showing that characters are inherited has gradually come to the fore,

and that is a comparison of identical twins with fraternal twins. Identical twins develop from the same fertilized egg, fraternal twins from two separate eggs ripening at about the same time. Identical twins are of the same sex (page 220) and have the same genes, except as mutations or chromosome aberrations may occasionally make them differ. Any quality which is inherited should therefore be the same in two identical twins, to whatever extent heredity is responsible for its nature. Fraternal twins may happen to be alike in some character because each one has the gene or genes responsible for that character; but they may differ with respect to it because they have different genes. Identical twins should therefore be on the average more similar in their hereditary characters than fraternal twins are. Conversely, any quality in which identical twins are more nearly alike than fraternal twins may safely be assumed to have a genetic basis.

When the mode of inheritance of a character is to be ascertained, the above types of evidence will not suffice. The best way of ascertaining whether a character is recessive or dominant, sex-linked or autosomal, dependent chiefly on one pair of genes or several is to have extensive family histories which may take the place of experiments. There are also statistical ways of judging recessiveness, which are applicable chiefly to rare characters, just as there are statistical tests of linkage. These methods will not be examined here, but the various degrees of definiteness of conclusions resulting from them will be constantly present.

Eye Color.—Color of eyes was referred to briefly (page 81) in discovering recessiveness or dominance of a character from a family history. Inheritance of eye color is somewhat more complex than was there suggested. The posterior layer of the iris contains a purple pigment, which shines through as blue if not concealed by anything in front. There is very commonly, however, a brown pigment in the front part of the iris. If this pigment is dense, the eyes are dark brown; if less dense, the colors are light brown, green, or gray. The quality of the color is also slightly affected by the texture of the iris.

Absence of the brown pigment (blue iris) is recessive to its presence (the various grades of brown). In general the lighter shades of brown are recessive to the darker ones, but there is much irregularity in this. Some investigators (Frets 1932)

have held the brown color to be due to two kinds of pigments and two pairs of genes, *Aa* for brown and its absence, *Bb* for yellow and its absence. In some races there are more dark eyes among women than among men, and it has been suggested that there is a sex-linked dark-eye gene in such races. This gene would have to be dominant to increase the number of dark-eyed females. An inhibiting gene, preventing development of brown and postulated to account for brown-eyed children of two blue-eyed parents, has already been mentioned (page 151).

There is a popular notion that eye color and skin color vary together, a notion which could easily be gained through failure to take racial origins into account. Correlation of eye color and skin color within a fairly homogeneous population seems never to have been adequately studied.

Finally, both the blue posterior and the brown anterior color of the iris may be missing, producing the pink iris of an albino. Although this condition may be part of general albinism, which affects also hair and skin color and is recessive to pigmentation, there is a form of it relating only to the eye. This latter pigmentless eye condition may accompany the usual color of skin and hair and is sex-linked and recessive.

Skin Color.—Color of skin plainly rests on a number of genes. Most races of men have some brown pigment, much or little. In addition, Negroes appear to have a yellow pigment which is readily concealed by heavy brown and which is recessive in crosses with whites, whereas Mongolians have a genetically different yellow which is dominant in crosses with whites. Inhibiting genes (the so-called dominant white) have also been postulated to account for a considerable number of instances in which mulatto children are darker than their parents. The inhibitor would be present in light parents, missing in the darker children. It should be pointed out, however, that children may be darker than their parents if the several color genes (of the same kind) are cumulative in their effect, after the manner of red color in the grains of wheat (page 151). This theory of Negro skin color has been held and corresponds well with the generally intermediate and very variable color of mulattoes, such as would be expected in blending inheritance (page 153).

Within the white race, differences in skin color may be much simpler. Family histories give the general impression that the

darker colors are dominant over the lighter ones, but lack of dominance or even blending could often be postulated. Lenz is of the opinion that these differences are not all due to pigment, but some to the structure of the skin (for example, his "rose-white" as contrasted with "ivory-white").

Distribution of skin color is sometimes irregular. Large patches of pigment are occasional, freckles rather common. The most striking examples of freckles are found in light-colored skins. The spotted condition is dominant over uniform distribution.

Albinism, or the complete absence of the usual pigments in the skin, is due to mutation of a general factor at the basis of all color, more specifically indicated with reference to hair color below. General albinism is recessive to pigment.

Color of Hair.—Several schemes of inheritance of hair color have been proposed. Basing his assumptions largely on what is known of coat color in other mammals, particularly rabbits, Lenz postulates the following system. There is a basic gene A without which there is no color, that is, aa results in albinism. All the laboratory mammals whose color inheritance is understood have such a gene, and it is fair to assume, since there are albino human beings, that man is no exception. In addition to this basic gene are the pairs Bb and Mm, which are the real pigment factors; B yields brown pigment, M black (melanism). A person whose phenotype is AbM or ABM is black-haired, while ABm is brown-haired.

Now, there are, Lenz concludes, a number of mutant alleles of A. He suggests first of all a, which as stated results in albinism, then a^1, a^2, a^3, and a^4. These multiple alleles represent increasing pigmentation, in the order named. The phenotype a^{3}Bm is called blond, while a^{3}bM is medium gray. Among these mutant alleles, the darker ones are held to be dominant over the lighter ones, as is generally true in the other mammals studied.

Another pigment, red, is produced in some individuals by the gene R. Its locus is likewise held to be the site of three or four alleles; nonred is r, while r^1 and r^2 produce larger amounts of red color. The complete range of color due to the various combinations of A, B, M, and R and their alleles may readily be imagined. They account for the diversity of hair color which man exhibits, and the proposed genes resemble those of other mammals. Whether man actually possesses these

genes could only be ascertained from extensive controlled breeding experiments.

The tendency of hair to whiten varies greatly in individuals and to some extent in races. Negroes become gray later than whites, and mulattoes are between these extremes on the average. Markedly premature whitening occurs in certain families (Fig. 124). In the one here shown it behaves as a dominant. It is sometimes objected that graying may depend on a hormone disturbance and hence not be genetic. Were the hormones themselves free from genetic control, that would be a valid objection. Where the same hormone abnormality, if that is

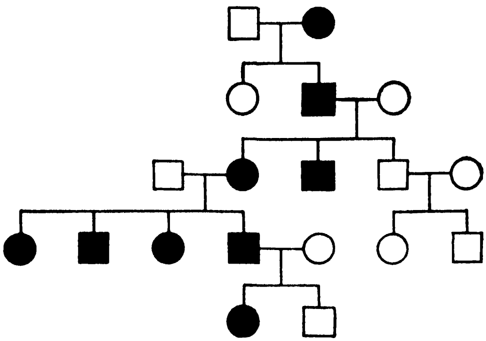


FIG. 124.—Five generations of premature whitening of the hair. The black squares and circles represent the affected individuals. In most of these the hair whitened before the age of 25 years. The character is here dominant. (*From Hare in Journal of Heredity.*)

what it is, occurs so many times in one family, as in Fig. 124, it is reasonable to attribute the peculiarity to genes.

Hair color is distributed unevenly in most people, as when mustache or beard is sandy while the hair of the head is dark, hair on the chest is different from that in the armpits, or hair on the front part of the scalp is lighter than that on the back of the head. In other mammals the agouti pattern is due to unlike placement of the colors on each individual hair, but man has nothing to compare with agouti. Nothing appears to be known about the inheritance of the distribution of human hair color.

Form and Distribution of Hair.—The form of the hair depends chiefly on its shape in cross section. If the cross section is circular or nearly so, the hair is straight; if an ellipse, the hair is wavy, curly, or woolly according to the degree of flatness (Fig. 125). It may even have a spiral shape. These features

are impressed on it as it develops from the follicle. Naturally there are many degrees of this flattening and many grades of curliness.

What passes for straight hair in the white races is recessive to the curly or kinky or spirally wound hair of Negroes and Hottentots. Within the white races, although straight is often said to be recessive to wavy or curly, it is probably more nearly correct to say that dominance is lacking. One proposed scheme of hair form includes two pairs of genes *Cc* and *Ss*, the former for curvature, the latter for spiral winding. Straight hair, in this system, is regarded as *ccss*; wavy hair as *Ccss*; curly *CCss*; and Hottentot spiral as *CCSS*. It is easy, however, to postulate more definite genotypes than our knowledge justifies.

Fig. 125.—Cross section of woolly (left) hair and straight hair. (*From Schokking in Journal of Heredity.*)

In the Mongolian races straight hair is genetically different from straight hair of European descent. Crosses with wavy-haired Hawaiians reveal the difference. While straight hair of the European types is recessive to the Hawaiian wavy, Chinese straight hair is dominant over it.

In monilethrix the individual hairs are alternately thick and thin, at intervals of about 1 mm. There is a tendency in hair of this sort to break off near the roots. It has appeared as a dominant character in some family histories, irregularly dominant in others.

Distribution of hair on the body is irregular and of somewhat the same pattern in different individuals. Yet there are racial differences, some races having abundant, others sparse, hair on the chest; some having heavy, others thin, beards. There are also individual differences in the extent of the mustache, and the shape of the beard as to the location of its principal hair. The heredity of these things is unknown. The principal specific indication that hair distribution is inherited is the pattern of its loss in baldness. In some it is the crown that first becomes bare, in others the front part of the scalp, in still others the whole top of the head (Fig. 126). These regions of baldness tend to be alike in various bald members of the same family.

Facial Characters.—In racial crosses, as in that between white and Negro, a broad nose appears to be regularly dominant over a narrow one. Within the white races, however, a high narrow nose is approximately dominant. Several genes are probably concerned, however, and some family histories are not quite so simple.

Thickness of the lips is undoubtedly genetic; but some have concluded thick lips are dominant, others that they are recessive, while Dunn in Hawaiian crosses finds hybrids intermediate. The genetic basis is not simple, apparently, or not always the same.

In some people, more often children, the skin of the upper eyelid is developed into a ridge or fold set obliquely across the

FIG. 126.—Pattern of baldness, spread over the whole top of the head. (*From Osborn in Journal of Heredity.*)

inner end of the eye opening. It is called the Mongolian fold, because it helps give the eye the appearance of sloping downward toward the nose. It does not have anything to do, however, with the real eye slope, as determined from the levels of the inner and outer angles. The Mongolian fold seems to be definitely dominant over the foldless type of lid, except in the Eskimo, where crosses with Europeans show it to be recessive. Perhaps the Eskimo lid fold is a different character.

A common difference in human ears is that the lower lobe in some is attached to the skin beneath, while in others it is free. While investigators have not all agreed concerning the mode of inheritance, most of them have found the free lobe to be dominant. Figure 127 is only part of a large family history assembled by Powell and Whitney (1937) which shows throughout the dominance of free lobes. A peculiar form of the ear which may be described as cup-shaped (Fig. 128) is reported by Potter

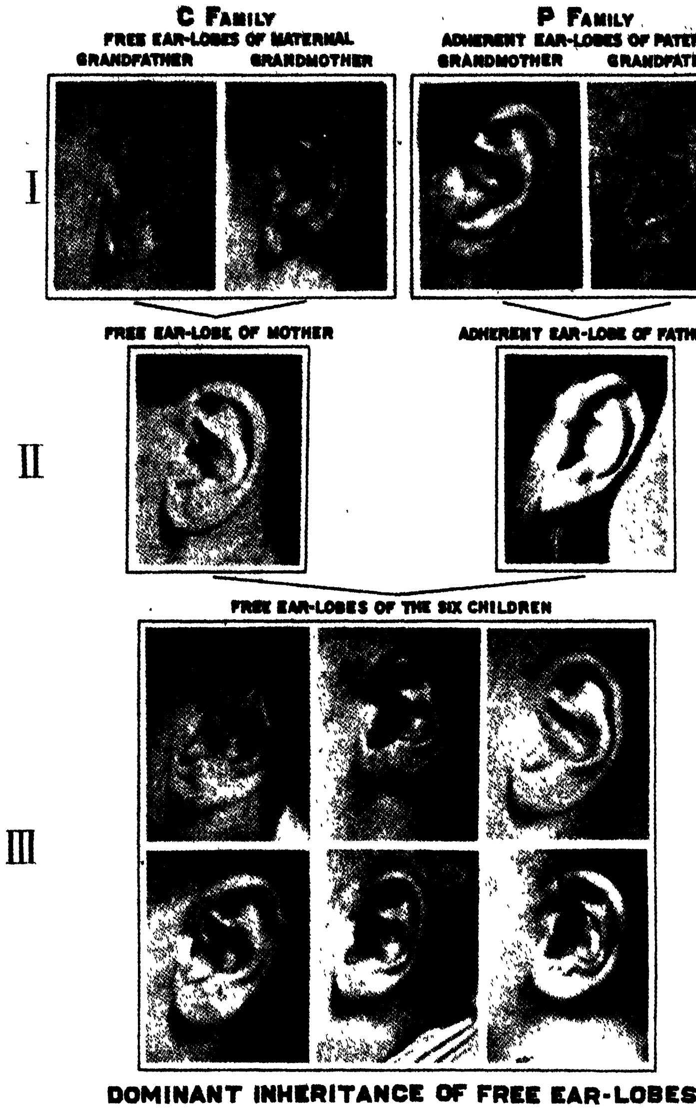


FIG. 127.—Inheritance of free and adherent ear lobes. (*From Powell and Whitney in Journal of Heredity.*)

(1937). It appeared in each of five successive generations and must be regarded as dominant.

The Teeth.—The time at which children cut their milk teeth is shown by twins to be partly inherited. Identical twins were found to cut them sometimes on the same day, sometimes a few days apart, and seldom more than two weeks apart; whereas of the fraternal twins studied none cut them the same day, and a great majority cut them more than two weeks apart. Differences in resistance to caries, however, are not very unequal in the two kinds of twins. This decay of the teeth is partly of dietary origin, but family histories show similarities of parents and children in this respect.

FIG. 128.—Cup-shaped ear, dominant over the usual type. (*From Potter in Journal of Heredity.*)

Size of the teeth appears to be inherited independently of size of jaw, for in crosses of Hottentots or Bushmen, whose jaw is small, with Negroes, whose jaw is much larger, the teeth of the hybrids did not conform to the size of the jaw. Crowding and displacement of teeth from their proper places in the row were a common result.

As a rather rare condition, the enamel of the teeth may be missing (Fig. 129). Both the temporary and permanent set are similarly defective. They lack the usual resistance to wear, and the teeth of a 20-year-old man were worn down to the gums. This defect was found in the direct line in each of six generations, hence is plainly dominant.

Teeth of one or more kinds are sometimes missing. Dahlberg (1937) reports the absence of six of the eight incisors in both temporary and permanent sets. He concluded that the defect is sex-linked and dominant. Other family histories on record show the upper lateral incisors missing in seven members of three generations or somewhat different anomalies in the incisors in various members of a pedigree.

These latter defects, relating as they do to the front teeth, may depend on some failure of the two halves of the jaw to

complete their growth and union in the middle line in the fetus. A smaller degree of the same failure may result in a slight gap between the middle incisors. This separation has been noted in families, and appears as a dominant character. An overgrowth of the jaws results in prognathy, which is regularly dominant.

The Jaws.—The tooth abnormalities just mentioned depend on a deficiency in the growth of the jaws, which may be much more extensive. The two halves of the upper jaw, as they grow toward one another in the embryo, may fall considerably short of uniting in the middle line. Harelip and cleft palate are the results. In some family histories these defects are nearly dominant, in some irregularly dominant, in some recessive and sex-linked. Whether there are autosomal recessive forms of the

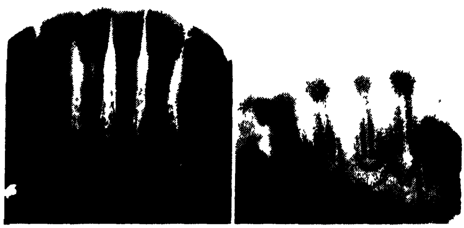


FIG. 129.—X-ray photographs of teeth without enamel (right); normal teeth at left. (*From Clark and Clark in Journal of Heredity.*)

defect is uncertain; this has been claimed, but the pedigrees in support of the suggestion have not satisfactorily proved recessiveness. Fortuyn (1935) interprets the data of various authors to mean that two pairs of genes are at work, one autosomal and one sex-linked. Reed (1936) finds harelip in mice to be due to many genes and believes that man follows a similar rule. The growth failure of the embryonic jaws is probably of several kinds, genetically different. Fortunately, by surgical treatment early enough, the defect can be largely removed; but the genes go on.

Abnormal overgrowth of the lower jaw, a condition known as acromegaly, is dependent on a diseased condition of one or more hormones but is held to have a genetic basis.

Head Form.—Because it is used so extensively by anthropologists as a racial character, the form of the head assumes some importance in human genetics. The shape is usually measured

by the ratio of greatest width to greatest length, a quotient which is called the *cephalic index.* A ratio of 0.72 is low, 0.86 is high. The former is that of a narrow or long head, the latter that of a broad or round head (Fig. 130). Between them and even beyond them in both directions is a finely graded series of indices.

The most decisive information about the shape of the head is that identical twins differ less than do fraternal twins. Some differences are due to distortion at birth, but these largely disappear. Identical twins then maintain on the average about the same difference in the index, or decrease it slightly, to the latest age adequately studied, which is about 25 years. Fraternal twins start with a difference about one fourth greater, and then increase it until it is roughly twice as great as the difference between identical twins of the same age. This contrast indicates the genetic basis of skull shape.

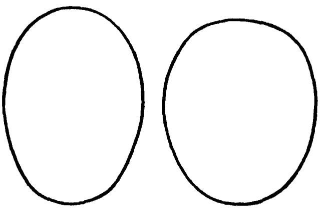


FIG. 130—Outline of head with low and with high cephalic index.

Discovery of the mode of inheritance is largely prevented by environmental influences. Growth in general is influenced by hormones, particularly that of the hypophysis. During the last generation or two, growth in man (as indicated by stature) has increased, probably because of increasing knowledge of hormones and vitamins. How has this growth affected the cephalic index? Deficiency of vitamin A in rats has diminished the length of the skull, thereby raising the ratio of width to length, and similar effects may well be produced in man. There are in fact some indications that increase of stature in man carries with it a lowering of the cephalic index. Despite these difficulties, theories of the mode of inheritance of head form have been proposed. Frets (1934) offers one suggestion to the effect that two series of multiple alleles are at work, the dominant member of one series favoring a long skull, the dominant member of the other series producing a round head. In conformity with this theory he finds in some family histories a dominant low cephalic index, in other pedigrees a dominant high index. The latter kind is more abundant. An alternative hypothesis by Frets is that height of the cranium is involved in its horizontal shape and that three pairs of genes are concerned.

In actual race crosses between broadheaded Hawaiians and narrow-headed Europeans, Dunn has found the broad head dominant. It is unlikely, however, that just one pair of genes is responsible. Different growth factors are presumably at work, and the ratio of width to length is not a good way of expressing their combined activity. Cephalic index is by no means a unit; the inheritance of each growth factor must be known before head form will be understood.

Microcephaly, abnormal smallness of the head, and a number of other cranial deformities carry with them some change of shape

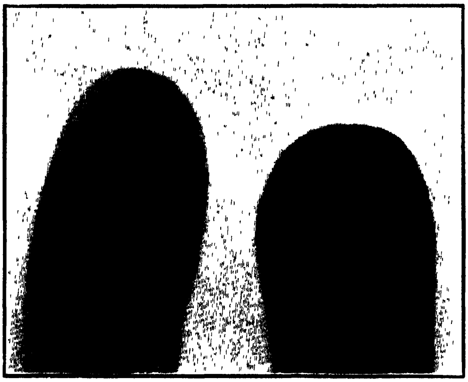


FIG. 131.—Radiograph of brachyphalangic thumb. Only the thumb was thus shortened, and only the terminal segment of it; one thumb (left) was normal. (*After Hefner in Journal of Heredity.*)

which has no bearing on the heredity of normal form. Some of these abnormalities are recessive, others dominant.

Bone Abnormalities.—Among the examples of human heredity cited in earlier chapters are several modifications of the skeleton of the hands and feet. Some of these are symphalangy, or fusion of the segments of the digits end to end without shortening, producing stiff fingers; syndactyly, fusion of the digits side by side; brachyphalangy, in which one or more phalanges are shortened (Fig. 131); and polydactyly, or extra fingers or toes. Brachydactyly or shortening of the fingers or toes as a whole, often to two phalanges per digit, is rather definitely dominant. These all probably rest on some irregularity in the rate of development of the affected parts.

A form of syndactyly which is particularly marked results in a division of the hand or foot into a fork, sometimes called a

"lobster-claw" (Fig. 132). Clubfoot, in which the deformed member is pointed downward, with toes in, appears in some pedigrees as recessive, in others dominant. This defect occurs in about one in a thousand and is found more often in men than in women. Congenital dislocation of the hip is of uncertain inheritance. Lateral curvature of the spine, once attributed to wrong posture or to rickets, has been found to extend over several generations as a dominant character.

Brittleness of the bones, due to scant development in thickness, occurs (if at all) in childhood but usually is reduced or lost at maturity. It is nearly dominant. The defect is often associated with a blue-gray color of the (usually white) sclerotic coat of the eyes, which is regularly dominant. It is suggested

FIG. 132.—"Lobster claw," a form of syndactyly. (*From Cook in Journal of Heredity.*)

that there is some common basis for the two characters. Deficient growth of the long bones at their cartilaginous zones results in dwarf stature, a condition which is sometimes plainly recessive, in other family histories dominant. Bone development is dependent on hormones, particularly those of the hypophysis, thyroid, and gonads, hence it is subject to possible nongenetic variation; yet in most instances the influence of the hormones is merely part of the genetic-physiological control.

General Growth and Size.—Not only the bones, but the body as a whole, grows or ceases to grow at the behest of hormones, particularly the three named as influencing the skeleton. So many things enter into growth and volume that a specific mode of inheritance for the whole process and its total product cannot exist. That size is inherited is shown, however, by studies of twins. In every ordinary criterion of size—chest measurement, weight, length of arms, length of legs, breadth between specific

points—identical twins have proved to be much more alike than are fraternal twins.

The shape of the body, whether slender or stout, is best measured by a ratio such as that of chest girth to height. It is commonly called body build. It is made up of different elements, of unknown number. Factors for stoutness are in general dominant or partially dominant in children, for the children of stout parents deviate less from the average of the general population than do the children of slender parents. In one study a group of stout women were found to have stouter parents than a group of slender women. In a number of family histories accumulation of fat appears to be dominant; but it must usually be related to the activity of some endocrine gland, and is subject to a considerable measure of environmental control.

Skin Defects.—Local expansions of the small blood vessels in the skin, resulting in habitual nosebleed when they occur in the mucous membrane of the nose, are due to a dominant factor. Pigmented spots or moles, also red swollen birthmarks, occur often enough in certain families to suggest dominant or irregularly dominant inheritance; but also they appear in lines that never before exhibited them and here must be environmental or possibly mutations.

In xeroderma pigmentosum the exposed parts of the skin develop pigmented spots and inflammation which usually lead to cancer, and the victim seldoms lives beyond his teens. Since this ordinarily precludes reproduction, the character cannot be expressed in a direct line, hence is not dominant. Though the mode of inheritance has already been suggested as following either the X or the Y chromosome (page 101), some have held it to be an autosomal recessive. It is a rare condition and perhaps may not be genetically always the same.

Epidermolysis bullosa is a tendency to form epidermal blisters in response to pressure, friction, or injury. It exists in several forms, one dominant, one recessive, and one sex-linked which may also follow the Y chromosome. Inflammation due to chemical stimuli is in general called eczema. Mostly it is probably a form of allergy, more particularly discussed in the next chapter. The mode of inheritance is not clear. Keratosis, a horny condition of the palms and soles, is dominant. In ichthyosis, the epidermis is covered with rough scales or horny plates.

A common form of it is dominant, another not clinically distinguishable from it is a sex-linked recessive, while congenital ichthyosis is autosomal and recessive.

Muscles.—Absence of one of the muscles of the forearm can be observed externally and has been traced through three successive generations as a dominant. Muscle dystrophy, a gradual wasting away of a certain group of muscles, is really due to a nerve defect. It is sometimes spread over decades, or may occur more rapidly. The victim often becomes almost helpless. It appears in some family histories as a dominant, in others recessive, or even partly as sex-linked. Atrophy of the muscles of the lower leg and the feet begins usually in children, but may extend in later years to the arms. The nerves to these muscles are also degenerate, and it is probable that the nerve defect is the cause of the muscle atrophy. In different pedigrees it has the appearance of a dominant, a recessive, or even a sex-linked recessive.

Another muscular deficiency due to nerve defect allows the eyelids to droop. It is called ptosis and is regularly dominant. Nystagmus, or rolling of the eyeball, though manifested by the muscles, is likewise a disease of the nerves. It is usually dominant, but in one pedigree it seems to be a sex-linked recessive and in still another an irregularly dominant sex-linked character.

Dimples, which depend in part on the arrangement of muscles beneath the skin, were long thought to be inherited—some said as a recessive, others as a dominant. The newer studies on twins, however, show that identical twins are only slightly more alike with respect to dimples than are fraternal twins. The environmental influence, much of it probably developmental, would seem to be large.

Hernia is to some extent due to the failure of a muscular wall to develop fully. The commonest type is that in which the viscera protrude through the inguinal canal. The testes descend from the abdomen through this passage late in fetal life, and then the canal should close. If it fails to do so, the viscera may sometime be forced out under stress of great exertion. From this origin, hernia is more common in men than in women; 3 to 5 per cent of men are affected. The transmission of this defect is practically that of a dominant, though a generation might appear to be skipped through lack of sufficiently strenuous physical labor to call attention to the rupture in some individuals.

Blood Vessels.—Varicose veins are swollen because of a weakness of the vessel wall. They are observed particularly in the lower leg because, in addition to the ordinary blood pressure, these veins must resist the hydrostatic pressure from the blood alone in the standing position. The weakness is generally dominant. A special form of this defect is observed in hemorrhoids, which are swellings of the veins at the end of the rectum. Sedentary habits and constipation are conducive to them.

Fingerprints.—The skin of the fingers is covered with a number of fine ridges which have so definite and distinctive a pattern for each individual that they are used as means of identification. The patterns are made up of three elements, namely, arches, loops, and whorls, combined in endless ways. Bonnevie has endeavored to show how these patterns are inherited. The number of the ridges depends on the thickness of the epidermis, the thinner it is the more ridges it bears. Bonnevie concludes that there is a general thickness gene *V* affecting all the fingers; that in addition there is a second gene *R* further determining thickness of epidermal pads on the thumb and first two fingers (radial digits); and finally a third gene *U* affecting such pads on the remaining two fingers (ulnar digits). Heterozygotes (*Rr*, *Uu*, and *Vv*) are held to be intermediate in epidermal thickness.

Further influence on the pattern is exerted by the shape of the fingertips. If a finger with thick epidermis is flat, the pattern consists mostly of arches. If the surface of the finger is strongly elevated, the discontinuous patterns (loops and whorls) prevail. There is more to the scheme but it is rather too complicated to present here.

CHAPTER XXIV

HUMAN HEREDITY: PHYSIOLOGICAL CHARACTERS

In this chapter are assembled some of the better understood or more important characters which are not primarily observed in structure. These qualities may be detectable only in activities, or the structural element may be obscure. In some of them there is a well-known structural modification, but the physiology is more conspicuous. In a few the structural expression may be the more conspicuous, but since the physiological basis of it is known it seems more fitting that the fundamental feature be emphasized by including them with other physiological qualities. And finally, there are some which could, as explained before, be equally well placed in either the physical or the physiological group. The mental characters, as physiological as any traits can be, are set apart in the next chapter.

Defects of the Eyes.—One of the common causes of blindness is cataract, an opacity of the crystalline lens. Often only a part of the lens is affected, and blindness is not complete. It appears first in advanced years in some of its forms, in children in other forms. Heat seems to favor its development, and furnace stokers have been more than proportionately susceptible to it. Some other unknown environmental (or modifying genetic) factors must also influence it, for, while cataract is in general dominant, it sometimes does not appear in every generation of a direct line (Hornback and DeGaris 1933). The cornea also has its opacities, one of which arises at puberty and gradually increases until vision is greatly impaired. These defects are simple dominants (Frykholm 1935).

Myopia or shortsightedness is due to a defect in the adjustments of refractive power. Refraction is due to curvature of the corneal surfaces and convexity of the lens, while length of the eyeball determines how much refraction is required. In myopia the structures are so shaped that at rest the vision is centered on nearby objects. More distant ones are not sharply

in focus. The popular idea that myopia results merely from close eye work seems to be mistaken, for the defect occurs only if the gene is present. Probably more than one gene is involved, however, for in different pedigrees it appears to be recessive or dominant. The opposite tendency, hyperopia or farsightedness, is usually dominant. When it occurs in children, it is frequently outgrown later.

Astigmatism is due chiefly to unequal curvature of the cornea in different directions. The image of a line in one direction may become a point on the retina, or the image of a point may become a line in another direction. At a given focus vertical lines may be distinct while horizontal ones are blurred. It is usually dominant, but family histories indicating recessiveness have been reported. In some families where astigmatism occurred repeatedly, the axis (direction of the line which forms a point image) was found to be the same in each individual.

In strabismus (squinting) the axis of the two eyes cannot be converged on the same point, at least at a suitable focal distance, owing to incoordination of the eye muscles. The eyes may be turned either inward or outward. The defect is ordinarily recessive. Operative correction is frequently possible.

Night blindness is due to a defect of the retina which is detected only because it prevents vision in twilight or similar weak light. It exists in several forms genetically and sometimes otherwise distinguishable. One form is dominant, one a sex-linked recessive, while a third which is correlated with myopia is an autosomal recessive. Retinitis pigmentosa begins in youth as a form of night blindness coupled with a narrowing field of vision. Eventually the sight is lost even in the middle of this field. Scheurlen (1935) regards it as an autosomal recessive; McQuarrie (1935) finds it sometimes a sex-linked recessive and perhaps also an autosomal dominant. The suggestion has already been recorded (page 101) that the gene may cross over between the X and the Y chromosome.

Sight by daylight is dependent on the cones of the retina, which are also responsible for color vision. In weak light it is the rods that function, and color is not detected. Night blindness is therefore due to a defect of the rods. When the cones are deficient there results a condition known as day blindness or (less appropriately) total color blindness. A day-blind person

can see by moonlight or other weak light, but is blinded by bright daylight. This condition is recessive, as indicated by the brief family histories in Fig. 133. Day blindness, despite its alternative name, has no relation to ordinary color blindness, already described as a sex-linked recessive character.

Deafness.—Hereditary deaf-mutism arises from a defect of the nerve of hearing or of the auditory centers of the brain, not from any abnormality of the ear itself. It is transmitted in recessive fashion. Unfortunately for the study of pedigrees the same defect results from several environmental causes. Meningitis and scarlet fever in early childhood may injure the inner ear and such children become deaf-mutes. Congenital deaf-mutism is often a result of syphilis.

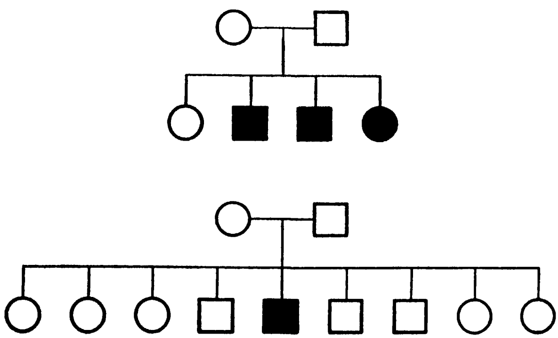


FIG. 133.—Pedigrees of day blindness.

Deafness due to a defect of the inner ear usually starts after middle age as a slight difficulty in hearing and becomes slowly or rapidly more pronounced, sometimes ending in total deafness. Anatomically two malformations of the labyrinth are known, hence there may be more than one kind of inner ear deafness. The various degrees of affliction may rest on these anatomical differences. Pedigrees quite regularly show the character dominant, although no homozygote appears to be on record. Dominance could therefore be lacking, but heterozygotes are distinguished from homozygous normal individuals.

In otosclerosis there is a disturbance of calcium metabolism affecting the growth of the bone in the neighborhood of the middle and inner ear. If this growth occurs in such a way as to reduce or close the connection of the middle with the inner ear, hearing is diminished. Although only two or three per thousand

in the United States are afflicted, the proportion in the families where it occurs rises to one-fourth or even one-half. Twice as many women as men exhibit the defect. Davenport holds that two genes are responsible, one of them sex-linked. Others have considered the character a simple dominant, or irregularly dominant, or occasionally recessive. There is some association of otosclerosis with brittleness of bones.

Taste.—Remarkable differences in the capacity to taste certain substances have been discovered. Many people do not taste phenylthiocarbamide at all, to others it is extremely bitter, to others salty or sour. Among those who experience a similar taste, the threshold of sensation is very different. Some people require nearly a thousand times as great a concentration as others in order to detect the substance (Blakeslee and Salmon 1935). Many other substances bring out similar though less striking differences in taste capacity. They show that the same person may have an acute sense of taste for one substance, a dull sense for another substance.

Taste "blindness," or the inability to taste a certain substance, was at first regarded as recessive. It is plain now that the matter is not so simple. If families in which both parents are tasters are compared as a group with those in which both parents are nontasters, it appears that there are more nontasters among the children in the latter group than in the former; but there are both tasters and nontasters in both groups. Were either the capacity to taste a substance or the lack of that ability a simple recessive character, one of these groups of children ought to be homogeneous, all individuals alike. The sense of taste must be more complex than it was first reported to be, and more refined tests will be needed to discover its mode of inheritance.

Allergy.—Strictly a physiological character is the sensitiveness of many people to foreign proteins. The irritating proteins may be those of pollen, or of foods, or of dust from hair. Hay fever, asthma, hives, eczema, edema, and migraine are expressions of this hypersensitiveness, and it has even been suggested that gall stones are due to allergy of the gall bladder.

There is dispute and uncertainty concerning the origin of individual examples of such irritation. Some have held that it is due to exposure to the irritating protein and that anyone may acquire any allergy. This view is scarcely in accord with

the greater prevalence of the response in certain lines of descent, and it seems necessary to conclude that even if allergy is developed by exposure this can happen only in individuals which have also the requisite genetic constitution. Allergy is more prevalent in cities than in rural areas, and more frequent in people of high intelligence than in those of lower IQ, but the meaning of these differences is unknown.

Most investigators have concluded that allergy is dominant or irregularly dominant. Skipping a generation might mean merely that a genetically allergic person had not been exposed to

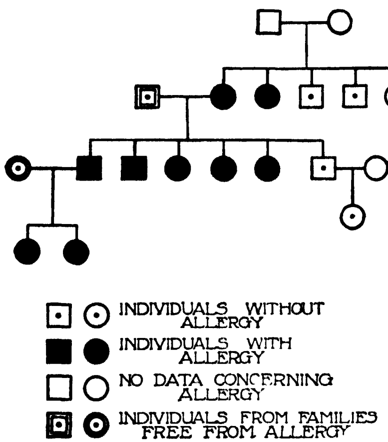


FIG. 134.—Pedigree of hay fever. (*Modified from Balyeat and Richards.*)

the protein to which he was sensitive. A brief family history with such an omission might even appear to indicate that allergy is recessive. Balyeat and Richards observed these uncertainties in a study of hay fever. In one of their pedigrees (Fig. 134) the conclusion that the character is dominant would be justified; but in another (Fig. 135) a generation is skipped at several places.

These same authors (Richards and Balyeat 1934) have also studied migraine. This is a recurring severe headache which may affect one side or the other, or some part of the head. It lasts several hours to a day and is probably caused by a swelling of the cerebral membranes. In some of the same pedigrees relating to migraine they found also asthma, bronchitis, eczema, hay fever, and urticaria (hives). It was observed that a parent

affected by one of these types of allergy might transmit another one to his children. They conclude that allergy is just one character, due to a single gene which is dominant. The various forms which the allergy takes may be due to environment or, perhaps, even to other genes not otherwise related to allergy. Though about twice as many women as men have migraine, this does not mean that the gene is sex-linked for the allergy may be transmitted from father to son. The excess of migrainous women is probably due to some influence of the sex physiology.

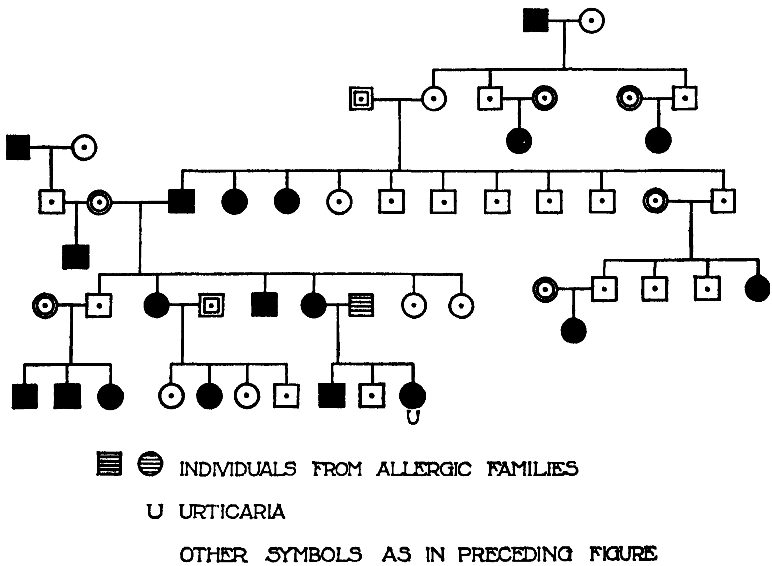


FIG. 135.—Pedigree of hay fever and other forms of allergy. (*Modified from Balyeat and Richards.*)

Blood and Blood Vessels.—The blood groups and hemophilia have already been described. One of the principal defects of the blood is pernicious anemia, a progressive reduction in the number of red blood cells or of their hemoglobin content. This malady occurs more frequently in some families than in others, and some pedigrees indicate that it is dominant, some are quite irregular. Paschkis (1936) believes it is dependent on two recessive genes, one of which acts on the gastric mechanism, the other on the bone marrow in which red cells develop. Patients are usually deficient in their gastric secretions, and there is some indication that this failure is dependent on a nervous dis-

order. Sickle-cell anemia is a rarer disease in which in drawn blood the red cells are sickle-shaped. It is accompanied by weakness, sometimes by ulcers and pains. The few known pedigrees indicate that it is an autosomal dominant character.

An important feature of blood vessels is the pressure which they maintain. Normally about 140 mm. in middle-aged people, this pressure may rise to 160, 180, or even 200 mm. with a corresponding increase in the burden on the heart. Reduced blood pressures are less common. High blood pressure could be regarded as a simple dominant character from most of the pedigrees, but environmental conditions (diet, mental strain) influence it considerably.

Closely connected with high blood pressure is arteriosclerosis, or hardening of the arteries. Most elderly people have some degree of this defect, and the occupational and other environmental influences are considerable. The indication that arteriosclerosis is hereditary is largely that in some families it has a tendency to affect the vessels of the same organs in different members—in some the brain where it leads to apoplexy, the coronary arteries of the heart in others, the vessels of the kidneys in still others.

The heart itself has some genetic defects. Inflammation of the valves is most often due to infection, but sometimes occurs so frequently in families as to make infection improbable as a sole cause. These pedigrees fit dominant inheritance best.

Metabolic Disturbances.—Though any physiological character is metabolic, medical writers have applied the term metabolic diseases chiefly to those relating to sugar metabolism, arthritis, and obesity. The last named has already been mentioned as a structural character because of its ease of observation. To the metabolic diseases are added, for this discussion, one pertaining to the kidneys.

Diabetes mellitus is an inability to utilize carbohydrates in the normal manner. The rejected carbohydrates appear as sugar in the urine and are thus eliminated. The disease appears chiefly in elderly people and is due to the failure of certain groups of cells (islands of Langerhans) in the pancreas to produce a normal hormone, *insulin*. The quantity of urine is increased, and the patient experiences great thirst and hunger and emaciation follows. Most family histories indicate approximately

recessive heredity, but some are irregular, and rarely they appear to show dominance. Men suffer from diabetes more than women do, but it is not sex-linked. Nondiabetic relatives of persons suffering from diabetes mellitus sometimes have more than the normal amount of sugar in the blood (Pincus and White 1934). These relatives may be future diabetics, but not all diabetes can be predicted in this way. A useful discussion of the variable heredity of the disease is given by Macklin (1933).

Diabetes insipidus is characterized by excessive discharge of urine, but there is no excess of sugar and no other unusual substance in the urine. The patient suffers great thirst. Family histories mostly indicate that the disease is a regular dominant, but there are exceptions in which heterozygotes do not show it (Levit and Pessikova 1936).

Renal glycosuria, in which sugar is likewise excreted in the urine, is not to be confused with diabetes. It is relatively harmless and does not rest on any progressive deficiency of the kidneys. It is inherited apparently as a dominant character.

Arthritis (gout) is inflammation of the joints, accompanied by an accumulation of urates in the tissues and of uric acid in the blood. The uric acid is probably not, however, the cause of the disease. Arthritis appears as a dominant character in most pedigrees but is irregularly so in some of them.

Susceptibility to Infection.—In infectious diseases, the principal feature of interest is a possible difference in the susceptibility or resistance of individuals to the infective organism. This difference is of especial importance when the causative germ is always or usually present. Resistance to tuberculosis has for this reason been the subject of much study. That this resistance is inherited is indicated by studies of twins. Of 39 identical pairs, 26 were alike and 11 were different in their relation to tuberculosis; of 69 pairs of fraternal twins, 17 were alike and 52 were different. This is a plain indication of heredity. Correlation studies of parents and offspring have led to the same conclusion. Both Pearl and Goring found this correlation (see Appendix) to be about 0.5. Since living in the same family might lead to infection, hence to correlation without genetic basis, similar correlation studies were made for husband and wife, in whom the genetic relationship would not ordinarily exist. Pearl found the husband-wife correlation to be 0.24,

while in Goring's study it was 0. The difference between 0.5 and 0.24, or between 0.5 and 0, is the correlation attributable to heredity. Pearl (1935) also found that 4.3 times as many people have tuberculosis when both parents are afflicted as when neither parent had it; and 1.6 to 1.7 times as many people have the disease if one parent has it as are diseased if neither parent has the infection. Only heredity seems adequate to explain all of these differentials.

These results are the more plausible because similar correlations in rabbits, where the infection with a standard bacillus could be carefully controlled, were calculated by Wright and Lewis, who found that 30 per cent of the difference in susceptibility was due to heredity, 10 per cent to age, weight, etc., and 60 per cent to environment.

Resistance to tuberculosis, as probably to most pathogenic organisms, is assuredly not a simple character. Lenz ventures the conjecture that the separate elements of susceptibility are recessive.

Susceptibility to Environment.—A number of defects have an environmental basis, but, even to these external agents, different individuals may respond differently. Rickets is due to a disturbance of bone development in early childhood. Already formed bones can be partially decalcified and become soft and capable of bending somewhat. Teeth are as susceptible as bones. Deficient sunlight and lack of vitamin D are the obvious causes of rickets, but under similar conditions some families suffer much more harm than others. Identical twins are more alike with respect to this defect than fraternal twins are. These results indicate inheritance, a conclusion supported by analogy with rats, in which rickets was more easily induced in some individuals than in others, and in which selection for 14 generations succeeded in establishing two lines differing in their susceptibility (Streeter, Park, and Jackson 1937).

A similar difference in susceptibility to goiter appears to exist but is slight. Goiter is an enlargement of the thyroid gland. It is favored by lack of iodine compounds in the food or water and is alleviated by very small quantities of iodine. The principal indication of heredity of goiter is the slightly greater similarity of identical twins as compared with fraternals. Cretinism, a dwarf condition which impairs mental development, is

in some way related to goiter. It occurs prevalently in regions where goiter is common and in certain families. The afflicted families usually have goiterous mothers, and it has been suggested that cretinism is transmitted by the cytoplasm of the egg (that is, as a non-Mendelian character).

Whether Mongolism is in any degree a family character has been debated, but the answer appears to be in the negative. This congenital defect involves a round shape of the head, large tongue and slanting eyes, and abnormal mental development. It certainly is not a dominant character, and the fact that cousin marriages do not yield Mongoloid children in undue proportion is against its being a recessive. Many investigators have noted that the defect occurs often in the children of older mothers. In view of these facts, it is suggested that some injury to the germ occurs as an accident of development. Rosanoff and Handy (1934), reporting some new twins involving Mongolism, summarize all published instances of twins as follows: 8 pairs of identicals in which both are affected, 36 pairs of fraternals with only one affected, plus 20 other pairs of unascertained type of which sometimes one, sometimes both, were affected. This is the chief evidence of inheritance of Mongolism. MacKaye (1936), however, finds a pair of fraternal twins (so judged because their eye colors and to some extent also their hair colors were different) who were both Mongols, showing that likeness of twins with respect to this abnormality can occur without genetic identity. Most authors continue to regard injury, in some way related to age of the mother, as the prime cause of Mongolism.

Longevity.—Length of life cannot be a single character, but it is so important in certain calculations and forecasts that it has received a great deal of attention. As a statistical entity, it must involve a number of immunities, which may be genetic. In some early studies of longevity Ploetz found that infant mortality (in the first five years) was only a third to a half as great in families in which one of the parents later reached an age of 85 or more as in families of shorter lived parents. Another early study, by Bell, showed that of the fathers of persons dying under 40 years of age, less than 21 per cent reached the age of 80; but of fathers of persons living to be 80 years old, 46 per cent reached 80 years. In families in which neither

parent lives to be 80, only 5.3 per cent of the children reach that age; if one of the parents lives to 80, 9.8 per cent of the children reach 80; and if both parents live 80 years, 20.6 per cent of the offspring attain a similar age. In the same family, the father's longevity was found to give a more reliable prediction of the children's age than did the mother's.

Many causes of reduced years are environmental and preventable. Fisher has calculated that if these preventable causes were removed, 13 years would be added to the average length of life. This is less than the amount added by having long-lived parents, indicating that heredity is really responsible in the latter group. Pearson has likewise employed mathematical methods to this problem, by the correlation procedure, and concludes that roughly two-thirds of longevity is due to heredity, one-third to environment.

Cancer.—Roughly one-tenth of all human beings who pass the critical years of early childhood develop some sort of malignant growth in later years. Though irritation of several kinds (X rays, chemical substances) helps to induce tumors, there are many indications that they have also a genetic basis. First is the frequency with which cancer appears in certain families. Little calculates that this frequency has not one chance in a million of occurring purely by accident, and Pearl describes a pedigree in which cancer occurs 200 times as abundantly as in the general population. There is a strong tendency for cancer to begin at about the same age in different members of the same family, and the chance that this could happen without heredity is very small. A number of identical twins have developed similar growths. It is frequently the same organ that is attacked in different members of a family. Thus among 258 breast cancer patients in an Amsterdam hospital, 76 had very near relatives afflicted with cancer, and 30 of these had breast cancer. Cancer of the stomach runs similarly through some families, intestinal cancer through others. Such likenesses must rest on a genetic foundation. Relatives of cancer victims are more often cancerous if the organ affected is breast, rectum, or stomach than if it is the liver or the uterus (Wassink 1935). Uterine cancer has been found more commonly in Gentiles than in Jews (Weir and Little 1934), and finally, cancer has been extensively studied in mice and some other animals and found to be definitely heredi-

tary in them. All this can be said while still acknowledging that environment is important.

When the mode of inheritance is sought, several theories are found, but there is little certainty. In glioma retinae, the evidence rather favors recessive genes. Some have supposed one or more recessive genes to be responsible for cancer in general, though certainly a single pair will not suffice. Others have regarded the gene as dominant, and there are family histories which could be simply explained in this way. It seems necessary, however, to assume multiple genes for cancer in general, and to regard the genes as different in different families or in different types of growth (Macklin 1932).

Nerve Defects.—Atrophy of muscles due to nerve degeneration has been described among the inherited structural characters. Several other nerve diseases may now be added. Spastic spinal paralysis, due to destruction of certain elements of the spinal cord, causes lameness and stiffness of the legs. One family history shows an unbroken line of descent for six generations, as if it were dominant, but more often the defect appears in the children of normal parents, which indicates recessiveness. Spastic paraplegia, marked by stiffness of the legs, irregular eye movements, strabismus or squinting and lowered powers of vision, is mostly recessive, though one pedigree indicates sex-linkage.

Friedrich's disease (hereditary spinal ataxia), due to degeneration of parts of the spinal cord, involves loss of coordination of movements of the limbs. Several extensive pedigrees indicate recessive inheritance. There is another form of ataxia which is dominant. Neither of them is to be confused with the ataxia due to syphilis.

Parkinson's disease, or paralysis agitans, is a fairly common condition arising in elderly people, in which the hands are in constant motion as in counting coins. Later a stiffness of the muscles develops, which may make the face resemble a mask, or which causes a stooping posture. Family histories indicate that it is a dominant character, though the fact that it appears only in elderly people could easily make it seem to skip a generation. Some who inherit the defect presumably do not live long enough to show it.

In Huntington's chorea or St. Vitus's dance there are likewise twitching movements of the hands, face, or other parts of the

body. These begin ordinarily between the ages of 30 and 50 and increase in later years. Mental degeneration accompanies them, and there is frequently a tendency to suicide. The many family histories agree (Fig. 136) in showing chorea to be a dominant character (see Sjögren 1935 for a recent study).

Myotonia or Thomsen's disease is present at birth in affected individuals. Muscles that have been at rest a long time enter into a state of stiffness or contraction which later disappears; or muscles may for hours at a time be quite incapable of contraction. Cold weather seems to favor these manifestations. Pedigrees indicate that the disease is a simple dominant character.

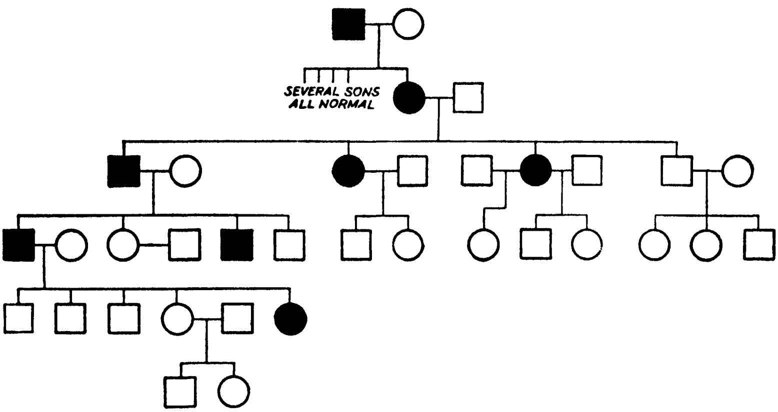


FIG. 136.—Pedigree of Huntington's chorea, which indicates that the defect is a dominant character. (*From Popenoe and Brousseau in Journal of Heredity.*)

Stuttering and other impediments of speech doubtless rest on some nervous defect, and there are indications that it has a genetic basis in part. Some families show several examples of it, as against about 1 per cent in the population as a whole. About half of the stutterers have stuttering relatives. Some have supposed that when children of stuttering parents also stutter, they do so in imitation; but some children of such parents have been removed to other surroundings and still stutter. The notorious Juke family included one branch with stuttering members in successive generations, indicating dominant heredity; but mostly the inheritance is not so clear. There is some indication that stuttering is related to left-handedness (Bryngelson and Clark 1933) and that it is more prevalent among twins, and twinning more frequent among stutterers (Berry 1937).

Left-handedness was found by Chamberlain (1928) to be more prevalent among the children of two left-handed parents (46 per cent) than among the children of two right-handed parents (2.1 per cent). Yet identical twins show little more similarity in the prevalent use of one hand than fraternal twins show. What determines handedness is not yet well understood.

CHAPTER XXV

INHERITANCE OF MENTAL CHARACTERS

Because of their complexity and the absence of unmistakable distinguishing marks in some instances, mental qualities are in general less well understood with respect to their heredity than are either the physiological or the structural characters. However, it is certain that many of them have a genetic basis even if the precise mode of inheritance has eluded discovery.

Feeble-mindedness.—The milder forms of mental defect are generally called collectively feeble-mindedness. There are many kinds and grades of it. Some are environmental, with syphilis and perhaps alcohol among the causes. In goiterous regions, feeble-mindedness occurs as part of the prevalent cretinism (page 271). The mental deficiency that inheres in Mongolism is probably due to age of the mother and accidental injury. Yet, after all these environmentally produced forms of mental defect are excluded, the bulk of feeble-mindedness must be attributed to genetic factors.

Some characteristic facts regarding heredity follow. Goddard found in a number of families in which both parents were feeble-minded that 470 of the children were feeble-minded, 6 normal. In other families in which one parent was feeble-minded and the other normal but shown by the pedigree to be heterozygous, 193 children were feeble-minded and 144 normal. In 26 families in which both parents were heterozygous, 83 children were normal and 39 feeble-minded. The ratios in these last two groups are near enough to 1:1 and 3:1, respectively, to suggest that feeble-mindedness is dependent on a single gene difference and that it is recessive. This assumption does not fit the first group, however, in which 6 normal children had sprung from two feeble-minded parents. There are many other family histories which similarly indicate that this mental deficiency is nearly a simple recessive, but with a few exceptions. Doubtless some of the exceptional individuals are illegitimate, but they can hardly all be explained in this way.

More probably feeble-mindedness depends on a number of genes and different genes in different examples. Normal functioning of the mind is made up of so many parts, derangement of any one of which should result in mental defect, that multiple-gene determination is to be expected.

Dementia Praecox.—One of the commonest mental diseases is dementia praecox or schizophrenia. Three-fourths of the inmates of insane asylums are committed for this defect. Often arising in the twenties, sometimes in earlier or later years, it takes many forms. Patients perform meaningless movements, or are subject to spells of rigidity, or have curious delusions. Their emotions and will are dulled, they lose interest in people and things, they are incoherent in thought and action. Their dullness and stupor may, however, be punctuated with periods of excitement.

Some have regarded dementia praecox as a dominant character, or perhaps irregularly dominant. Others have thought it necessary to assume two genes, either alleles or at different loci.

Other Insanity.—On the basis of statistics from institutions in New York State, about 4.4 per cent of all females and 4.7 per cent of all males are treated for mental disorder at some time in their lives. The actual number of psychotic individuals is presumably larger. About 10 per cent of the affected males and 20 per cent of the females are treated for manic-depressive psychoses. This is probably a group of different but related disorders. Patients are subject to periods of severe melancholia and strong excitement, sometimes alternately.

Pollock, Malzberg, and Fuller (1934) think there is little evidence of a genetic foundation for manic-depressive insanity. They say, for example, that when one member of a family exhibits one of these psychoses, the remaining members of that family are no more likely to have some mental disorder than is the general population but that, when they are disordered, these brothers and sisters of a manic-depressive are more likely to show manic-depressive insanity than some other psychosis.

Twin studies, however, speak strongly for inheritance. In identical twins 31 pairs were concordant, 2 discordant; while in fraternal twins 1 pair was concordant, 13 discordant. Those who find manic-depressive insanity inherited have mostly regarded the elements which contribute to it as dominant.

When insanity is considered a unit, without respect to the type of disorder, no conclusion as to mode of inheritance is to be expected. The pedigree in Fig. 137 shows presumable transmission but no definite rule for it.

Amaurotic Idiocy.—Children born apparently normal begin sometimes to lose their sight, become crippled, and recede in mental development in their early childhood years. The disease results from degeneration of nerve ganglia and the retina of the eye and regularly ends in death in early life. It is said to affect almost exclusively Jewish families and is a simple recessive character.

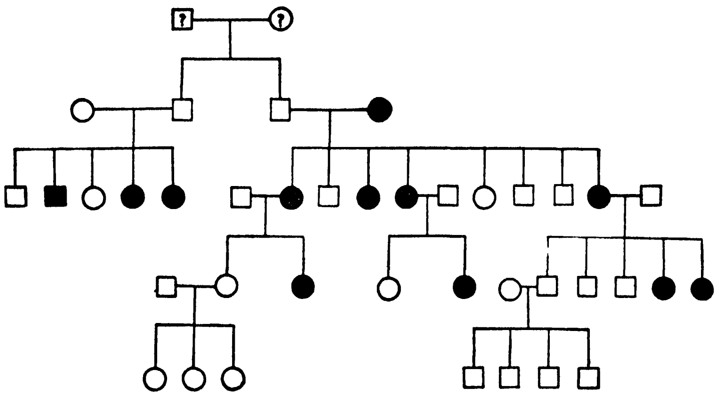


FIG. 137.—Pedigree of insanity, indicating that the defect is inherited but leaving the mode of inheritance in doubt. Black, insane; shaded, peculiar, queer, highly nervous; white, normal. (*From Hanson in Journal of Heredity.*)

Epilepsy.—This defect is manifested by spells of unconsciousness and peculiar muscular spasms. There is considerable association of epilepsy with feeble-mindedness in the same family histories, and it is difficult to avoid the conclusion that they have some basic factor in common. Conrad (1935) finds epilepsy nearly twice as common in twins as in the general population, and twenty times as common in identical as in fraternal twins. About 10 per cent of epileptics come from epileptic parents, and about 10 per cent of the children of epileptics are likewise epileptic. While these facts all point to heredity, the mode of inheritance is uncertain. There are probably recessive genes, but also dominant ones, which contribute to it.

Crime.—Crime is assuredly not a single character, and what constitutes a crime depends on the laws under which the individual lives. Also whether an individual is criminal, or rather how criminal he is, is partly determined by his environment. Yet it would be easy to overrate the influence of these nongenetic elements in crime, for a person whose mental qualities lead him to disregard the rights of others is well on the way to crime no matter what the laws are. Furthermore the facility with which a person gets into a crime-breeding environment is often one of his most distinctly hereditary characteristics. Genetic constitution not only helps to choose its environment, it helps to make it. This is one of the chief weaknesses of the view so often held that society itself is responsible for the crime in its midst. While admitting that the penal system may help to perpetuate itself by making itself necessary, one need not dismiss the hereditary element.

A few of the pertinent facts follow. Some of the mental defects known to be inherited naturally lead to crime. The cold cruelty and thoughtless violence that often go with schizophrenia easily have criminal consequences. Psychopathic individuals easily conceive that they have suffered a great wrong and are led to crime out of conviction that it is just. Epileptics are guilty of crimes of violence more often than is their numerical share. Feeble-minded people do not foresee the consequences of their acts, and are led to follow their desires. One study of 470 youths who came into conflict with the law showed 30 per cent to be distinctly feeble-minded, while 70 per cent were of lower intelligence than the average for their age. Mental tests have shown that repeating criminals have on the average a low IQ. In a study of prostitution in Copenhagen, Kemp (1936) found that only 29.4 per cent of the more than 500 women included were mentally normal. Their families showed a high incidence of psychopathy, insanity, oligophrenia, alcoholism, criminality, and suicide. There are, of course, some types of crime that require high intelligence.

The criminal records of twins are of interest. One study showed that in 10 pairs of identical twins both had a criminal record while in 3 pairs only one was criminal; and in 2 pairs of fraternal twins both were criminal, in 15 pairs only one. A number of these twins were repeaters. Another investigation

indicated a difference in the same direction between identical and fraternal twins, but a difference less striking. In this study the first offenders made up a larger share than in the former one, which may account for the smaller difference. Two of the pairs of identical twins were reared apart since youth, yet each had a record of repeated arrests and convictions.

Some penologists have carried the idea of predestination to crime so far as to hold that criminals may often be recognized by their physical traits. Were this true, the environment could perhaps be adjusted for such individuals in a way that would reduce their criminal performance, or such criminals when apprehended could be given different treatment. Unfortunately, there seems to be no physical character that serves as the mark of Cain. Careful measurements show that the heavy jaw, asymmetrical face, lobeless ears, sparse facial hair, and shifting eye, which have been proposed as signs of criminality, do not occur in criminals enough more frequently to be of use. Forgers have on the average poorer health than other criminals, and crimes of violence are done by people stronger than the average; but no satisfactory forecast is possible from any physical or physiological quality.

Notorious Families.—Because of the hereditary nature of some elements of criminal tendency, families tend to exhibit it frequently if they exhibit it at all. A number of notoriously criminal families are on record. One of the earliest of these is the so-called Juke family in New York State, carefully studied by Dugdale up to the year 1875. The name Juke is fictitious, but the real name is known, and the family history was brought down to 1915 by Estabrook. In the interval between these two studies, the family had changed little except in numbers. Of nearly 2100 individuals (three-fifths of whom were still living in 1915) belonging to this family, 378 were prostitutes, 181 victims of alcoholism, 170 paupers, 129 otherwise dependent, 118 criminals, and 86 kept houses of ill-fame. Approximately half of the family are feeble-minded, and much more than half of the criminal part of the family are feeble-minded. Indeed, by Estabrook's classification, all criminal members known to him were rated as mentally defective. All these people are descendants of one woman, named "Ada" in the records, who was herself a harlot, and the son of a backwoodsman of Dutch

descent. Little has been ascertained directly about the mental qualities of this pair, but it is likely that both were defective. These two people, through their descendants, without counting any indirect losses, have cost the state of New York millions of dollars.

Another such family was later studied by Goddard and described under the name Kallikak. The progenitor of this family, himself of respectable though not prominent ancestors, in Revolutionary times became the father of an illegitimate child by a feeble-minded girl, and later married a cultured woman of good family by whom he also had children. Two lines of descendants were thus begun, one of which has been almost wholly normal, the other including many feeble-minded and some other defectives.

The Tribe of Ishmael, studied first by McCulloch and later by Estabrook, furnishes additional examples of what is presumably mental defect running through general families. This notorious group is not of a single relationship, but includes perhaps four hundred families, of which the Ishmael family was the largest and worst. An early record is that of John Ishmael, who migrated from Kentucky to Indianapolis in 1825, and thereafter made "gypsying" trips annually, returning to the vicinity of Indianapolis for the winter. Other families with similar wandering proclivities followed the same route and stopped at Indianapolis because of the generosity of the inhabitants. Central Indiana thus became the headquarters of numerous families having similar qualities and receiving collectively the designation Tribe of Ishmael, though not of a single kinship. They have since spread more or less into the surrounding states and number almost certainly more than ten thousand. The characteristics of these people are shiftlessness, gypsying, petty thieving, begging, and sexual immorality, including prostitution and polygamy. Without much doubt these traits are at bottom due to feeble-mindedness or other mental defects.

Similarly notorious are the Zeros of Switzerland, the Wins of Virginia, the Hill Folk of Massachusetts, and the Nams of New York. To describe them would be largely repetition. The fundamental cause of their peculiarities must be in each of them widespread mental deficiency.

Intelligence.—On the credit side of the genetic ledger no item is of more importance than intelligence. Mental defects have already been discussed. What is now to be considered is the variety of abilities within the normal range. These are harder to follow than are the unusual capacities, yet there is unmistakable general evidence that they are inherited. Schuster and Elderton found that of "pass" students at Oxford, 20 per cent of the fathers had taken first or second honors; while of honor students, 42 per cent of the fathers had taken first or second honors. Woods, in a study of European royalty, found the similarity of parents and offspring, with respect to ability, to be about twice as great as that between grandparents and grandchildren.

Mental tests have been used with school children, and have shown that of 41 superior children only 2 lacked a near relative who was also superior. Identical twins furnish the usual valuable information. The first critical examination of such twins was made by Muller. The twins in question (Fig. 138) lived in very different environments, although in families of the same general social rank. Comparison of their physical likenesses and differences indicated, by a statistical method, a probability of 386:1 that they were really identical twins and should therefore have identical inheritance. Intelligence tests resulted in strikingly close ratings—153 and 156 with the army alpha test, and 62 and 64 with the Otis advanced test. These are very superior ratings. In other tests, designed to reveal will, temperament, emotion, social attitude, etc., considerable differences were revealed which are fairly attributable to differences in training and other experience. Later studies of identical twins by Newman did not lead to such clear results. Using fingerprints and other physical characters to determine which twins were identical, he found that the mental equipment of some of those regarded as identical might be very different. One pair, described rather fully, lived together until two years of age, after which one went to Canada, the other remained in London. Their physical likenesses indicated a probability of about 2000 to 1 that they were identical. Eleven tests were applied to them, including the Stanford-Binet, Thurstone psychological, Otis self-administering, International (Dodd), Stanford achieve-

ment, and Downey will-temperament. The twins were quite different in nearly all cases. One may reasonably inquire, in view of the differences, whether the twins were really identical, since similarities were the basis of declaring them such. The fingerprints prominently used in studies of identical twins

FIG. 138.—Identical twins who were separated at the age of eight months and have been together an aggregate of ten months since that time. One was trained in a business college and has held numerous secretarial positions; the other has been a teacher, is married and has one child. Their physical likeness is obvious. Their mental likeness has been tested by Professor Muller (see text). (*From Popenoe in Journal of Heredity.*)

are shown by Cummins to be not always alike in twins possessing a single chorion at birth, a feature regarded as usually diagnostic of such twins. Furthermore, Gates raises the question whether determination of identical twins by mere likeness in many respects is valid. He cites a set of triplets which might easily be regarded

as identical, but whose other brothers and sisters, of different births, were also much alike, and concludes that the likeness was due to homozygosis of the parents. One would like also to know what results would be obtained by administering the various psychological tests to identical twins who had lived together all their lives.

Banker (1931) devised a "student's ability index," derived from school marks, chronological age, and educational age of the individual and the group to which he belongs, and used it to study inheritance of ability. After obtaining the SAI for many individuals and their parents, he grouped them into low, medium, and high. The six possible matings between parents of these three grades were then separately studied, and it was found that in general as the ratings of the parents rose the indices of the children steadily increased.

How intelligence is inherited is not understood; few have attempted to discover a definite method, owing to its presumable complexity. Hurst (1934), from a consideration of the numbers of people of the various mental grades, has proposed a scheme involving six pairs of genes which he calculates will produce the observed distribution. First in his scheme is a dominant gene *N* for mediocrity. In the presence of this gene other genes are of no effect. With *nn*, which permits atypical mental development, either good or bad, modifying genes (*Aa*, *Bb*, *Cc*, *Dd*, *Ee*) determine the result. These modifiers are cumulative and lack dominance. One gene of each of these pairs favors ability, the other the reverse. Hurst estimates that the random combinations of these genes would provide the observed numbers of individuals of the various grades of intelligence, but no attempt has been made to determine the genotypes of individuals in any family history.

Musical Ability.—The classical evidence of the inheritance of musical ability is the family relationship of many musicians. A favorite pedigree for this purpose is that of Johann Sebastian Bach which, because of errors in all earlier accounts of it, has recently been carefully re-examined. The new family history may be summarized as follows. In six generations, taking into account only men (because of the limited opportunities for women) and omitting all not old enough to have demonstrated their capacities, this pedigree includes, besides Johann Sebastian

himself, 29 professional musicians, 16 others who were composers, 2 known to have musical ability but who were not professional, and 7 who are not known to have had any special musical gifts. This list is complete, no men have been omitted. Similar pedigrees have been assembled for Mozart, Beethoven, Brahms, Schubert, Liszt, and Weber, each through three or more generations and each including many more musicians than the general population can boast.

If musicians married at random, among musicians and nonmusicians in proportion to their numbers, the continued appearance of musicians in a pedigree in every generation would indicate that musical ability, of whatever it consists, is in a high degree dominant. There is, however, much selective mating; musicians marry musicians. Even after making allowance for this concentration of musical genes, it still seems likely that the elements of this ability are largely dominant. A celebrated living pianist, whose wife is also a professional musician, has a son who is quite deficient in this respect, a result which would readily follow from dominance of musical ability. Mjøen observed in 114 families that two nonmusical parents always had nonmusical children, while two highly gifted parents might have children less capable than themselves.

It is not profitable, however, to try to fix dominance or recessiveness of musical ability as a single character. It is obviously made up of sense of pitch, consonance or dissonance, intensity and rhythm, tonal memory, feeling, imagination, and the like. Psychological tests devised by Seashore have been used to measure some of these separate elements, and it is found that they may be fairly independent (Stanton 1923). A person may have a good sense of pitch with a poor memory for tones, or a good sense of intensity without a good recognition of consonance. The results of these tests are not appreciably affected by musical training (Stanton 1934), hence are to be regarded as measures of ability, not achievement. Even these single abilities have not been shown to be inherited in any clear fashion, as children may be either superior or inferior to or intermediate between two dissimilar parents. Inheritance of musical ability is therefore quite complicated.

Other Special Aptitudes.—Painting and sculpture have likewise their celebrated families. Titian was one of nine artists in

one general relationship, and the historical painter Feuerbach was similarly related. Mathematics is also a family accomplishment; the Bernouilli family included at least eight important mathematicians. This ability may concern chiefly form (geometry) or logic (algebra, etc.), and one investigator concluded that divisions of mathematics tended to run separately in families. This same student held that the single components of mathematical ability were probably mostly dominant and autosomal.

Similar claims have been made for technical skill, perhaps correctly. But since opportunities to use technical ability are mostly in commercial and manufacturing establishments, to which family connections often provide the entree, the evidence is somewhat weakened. Geniuses tend likewise to accumulate in families, as is easily recognized in the pedigree of the Darwins and Galtons, and is less strikingly true in a number of others. In one study of genius it was found that 80 per cent of the fathers of 282 eminent men were of nobility, professional men, semiprofessionals, higher businessmen, or members of the gentry. Here again family connection sometimes spells opportunity, but this consideration merely weakens the evidence without destroying it.

Attempts have been made to relate character and intelligence. While these qualities seem on the whole to go together, there are exceptions, and conclusions regarding their connection are vitiated by lack of a suitable objective measure of character. Doll (1937) has similarly studied inheritance of "social competence," a quality which he measured by reference to a series of statements of what people normally do at various ages. The tests were taken either in person or *in absentia* on the testimony of a third party. Three family histories are shown, one superior, one borderline, one subnormal. The social quotients (SQ) are fairly consistent within each family and are closely related to the intelligence quotients (IQ). Such studies probably have a long way to go, however, to have much independent evidential value.

CHAPTER XXVI

PRACTICAL APPLICATIONS OF HEREDITY

The economic uses of heredity are found in the improvement of agricultural crops and domestic animals. Certain qualities are desirable in plant and animal products, and varieties differ with respect to them. Quantity is also important, and different lines of descent are found to be unequal in yield. The breeder seeks to accumulate and accentuate these desirable properties. The social applications are mostly in improvement of the human race itself but to some extent in the settlement of legal questions.

Method of Improvement.—The method of improving varieties has always been that of selection, choosing as parents for each generation those individuals which will presumably transmit the desired qualities. In the choice of these individuals two standards have been used. In one method of selection, the qualities of the individual are chiefly considered. Any animal or plant which has, or shows the nearest approach to, the desired attributes is chosen for breeding. In the other plan, the breeding individual's own qualities are considered less important than the qualities of its relatives. Parents of each generation are taken from families having the preferred characters in high degree. Each method has had its advocates. Selection has sometimes progressed under one system, when it failed under the other, as when Pearl succeeded in raising the egg-laying capacities of a flock of poultry by the family-performance method after the individual-performance plan had failed.

The most certain way of using family performance as the criterion is the progeny test. A parent that actually produces a high percentage of, or a near approach to, the desired qualities in its offspring is better than one which comes of good family but may individually transmit less than the family standard. Lush (1935) has made a biometric study of the progeny test and individual performance as indicators of breeding value and concludes that the progeny test is the more valuable when large numbers of offspring are available, but not for so few as five;

when the only thing causing the progeny to resemble one another is that *one* parent is common to them; and when the character is only slightly hereditary. With respect to the first of these requirements—large numbers of progeny—plants and animals are often very differently situated. Most plants are easily reared and are capable of producing many offspring, whereas the larger domestic animals are expensive to keep and reproduce slowly. As a consequence the selection of these larger animals has mostly been based on individual qualities; and yet when a bull begets a prize-winning calf, one conforming closely to the standards of the breed, its value as a sire promptly rises. On the whole, the progeny test is available for plants, less so for the common animals except perhaps poultry.

Resistance to Disease in Plants.—One of the chief elements of yield in farm crops is the ability of the plants to resist disease. Wheat rust, a fungous disease, exists in more than 40 physiological forms, to which the varieties of wheat react differently. Mains finds that Malakoff wheat is resistant to form 12 but susceptible to form 5, while another (unnamed) variety reverses these relations. The resistance of Malakoff to form 12 is dominant over the susceptiblity of other varieties to form 12, but the resistance of the unnamed variety to form 5 is recessive. Resistance of Webster wheat to form 5 is neither dominant nor recessive to susceptibility. Resistance and susceptiblity of Malakoff to form 12 differ by just one pair of genes, although in Kanred they differ by several. According to Aamodt the gene for susceptibility to form 1 in Marquis wheat, that for moderate resistance in Kota wheat (Fig. 139), and the gene for immunity in Kanred constitute a series of multiple alleles. One of the immunity genes used in wheat varieties comes from emmer, a relative of wheat (Clark and Smith 1935).

Resistance of oats to all forms of its stem rust except form 6 has been found in one or more varieties of the grain. Resistance differs from susceptibility by just one pair of genes, except in the reaction to form 4 which is governed by two pairs. Resistance is dominant in the single-gene crosses. Bond oats, which is resistant to crown rust, has been crossed with Iogold, Anthony, and others which are resistant to stem rust, and the offspring show various combinations of resistance (Murphy, Stanton, and Coffman 1936). Here the reactions are not so simple.

Smut, both loose and covered, has been studied in crosses of oats varieties and both were found to be dominant with a ratio of 3 resistant to 1 susceptible in F_2, indicating the effect of only one pair of genes (Reed 1934).

Rust resistance in beans is due to one pair of genes and is dominant over susceptibility (Wingard 1933). Resistance of beans to the mosaic virus has already been mentioned (page 212) as partially non-Mendelian; it is due to different properties of the cytoplasm in different varieties.

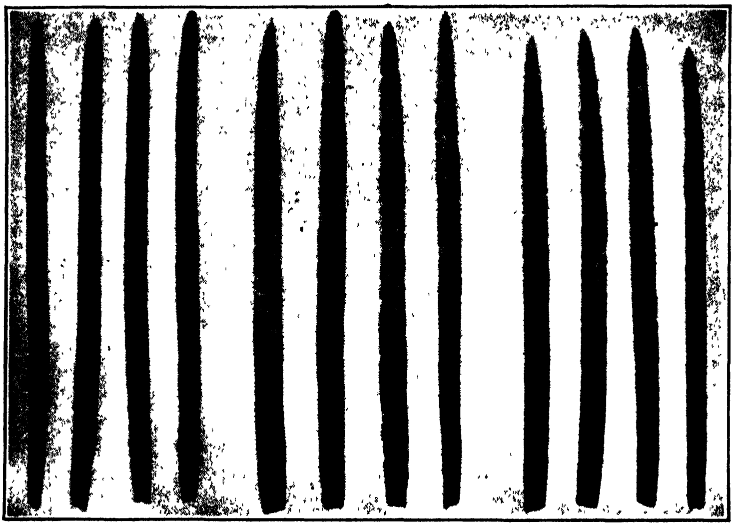


FIG. 139.—Differences in resistance of wheat to black stem rust, race 27. Three F_3 families derived from a cross of Marquis × Kota; at the right a susceptible family, in the center a somewhat resistant family, at the left an immune family. (*From Hayes and Aamodt.*)

Tobacco is susceptible to its mosaic virus in different ways in different varieties. Some varieties merely exhibit the mosaic disease, while in some there is an additional susceptibility gene which causes a necrotic degeneration (Holmes 1937). Resistant varieties of tobacco have been imported from South America in an attempt to introduce resistance into the favored varieties in the United States.

Resistance to anthracnose differs considerably in raspberries. The variety known as Quillen (Fig. 140) is one of the most resistant, and red varieties are more resistant than black ones. Several pairs of genes are concerned with these distinctions.

Varieties of cabbage differ greatly in their resistance to the "yellows," a disease caused by a fungus which develops best in hot weather. The Copenhagen variety is very susceptible, whereas Marion Market is nearly immune (Fig. 141). The heredity of this resistance is being determined.

Fig. 140.—Resistance to anthracnose in raspberries. *A*, Cumberland, a susceptible variety; *B*, hybrid of Plum Farmer × Cumberland, moderately susceptible; *C*, hybrid of Honey Sweet and Plum Farmer, rather resistant; *D*, Quillen, immune. (*From Colby in Journal of Heredity.*)

The physiological properties through which immunity is attained are mostly unknown. Marañon finds that evening primroses (Oenothera) immune to mildew contain larger amounts of tannin and water-soluble acids, while the susceptible plants have more than the average content of nitrogen and ash. In

wheat, Sax concludes that the varieties with the smaller numbers of chromosomes have in general the greater immunity.

Resistance to Disease in Animals.—Much less definite is the knowledge of inheritance of resistance to disease in animals. The resistance of poultry to *Salmonella pullorum*, the bacterial germ of white diarrhea, can be increased by selection, and crosses show resistance to be dominant (Roberts and Card 1935). Resistant birds have more red cells in the blood, susceptible fowls more white cells, but these differences may have nothing

FIG. 141.—Copenhagen cabbage, susceptible to the "yellows," at left; Marion Market, resistant, at right. (*From Reinking in Farm Research, N. Y. State Agr. Exp. Sta., Geneva.*)

to do with the resistance. Susceptibility to typhoid in fowls was reduced from 85 per cent to 10 per cent in five generations of rigid selection (Lambert 1933), but even then considerable genetic variability remained in the flocks. The mode of inheritance was not discovered in this study. Frateur concluded that resistance to diphtheria in poultry is due to a single pair of genes, but the numbers of fowls observed was too small to justify so definite a conclusion.

Rabbits differ considerably in their resistance to the bacterium causing abortion, and Manresa was able to produce by selection nearly true-breeding strains of susceptible and resistant

animals. He tentatively concluded that the difference is governed by one pair of genes and that resistance is dominant or partially dominant.

Many other experiments with resistance to infection have been conducted on laboratory mammals, particularly rats and mice, but these animals are not of direct economic importance in the same sense as poultry and rabbits. They confirm by analogy, however, the expectation that such resistance will be found to be genetic and that it is probably Mendelian.

The question has been raised whether resistance may not be more dependent upon the mother than upon the father and so be attributable to cytoplasmic or (in the mammals) developmental influence. In a number of experiments this possibility has been tested by making two series of matings, in one of which only males were selected, in the other only females. The results were essentially alike, indicating that resistance is transmitted nearly equally by the two sexes.

For an excellent discussion of the inheritance of disease resistance in animals, reference may be made to the work of Lambert, cited above.

Resistance to Pests.—The variety of wheat known as Dawson is resistant to the Hessian fly, only 0.4 per cent of the plants being infested. Poso and Big Club, however, are very susceptible, with 97 or 98 per cent of infestation. The insects are unable to complete their life cycle on the resistant variety. In crosses of Dawson × Poso and Dawson × Big Club it appears that resistance is dominant. The F_2 generation consists of resistant and susceptible in the ratio of 15:1, indicating that two pairs of genes are involved, and that resistance is provided by either or both of the dominant genes (Cartwright and Wiebe 1936). Whether they are duplicate genes (page 157) or not has not been ascertained; but since wheat has 42 chromosomes and is believed to have come from a species with 14 through a multiplication of the chromosomes, it is quite likely that the genes are duplicates.

Resistance of corn to the European corn borer (Fig. 142) has been tested in hybrids, rather than in pure strains, to take advantage of hybrid vigor (see below). Some years ago it was suggested that resistance of Mais Amargo, a South American variety of corn, is a simple genetic character recessive to the

susceptibility of Michigan strains. Recent studies in Ohio, however, indicate that infestation is greater on the early-maturing and taller plants, which may be dependent on factors having no relation to real susceptibility (Meyers *et al.* 1937). High productivity seems to be the best means of combating the borer, at least so long as the degree of infestation is as low as at present.

Grape vines of some varieties are attacked at the roots by insects known as phylloxera. Since no way of destroying the insects could be found, immunity was sought. Resistant varieties were known, but they were very inferior in the quality of their fruit. While the heredity of this resistance has never been determined, hybridization would doubtless have produced eventually a resistant variety with suitable fruit. It would have been a long process, however, and a quicker method of saving the vineyards was employed, that of grafting stocks of desirable varieties upon the roots of resistant ones.

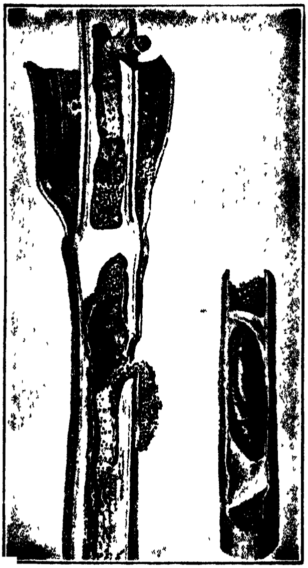


FIG. 142.—The European corn borer. (*From Snodgrass, U. S. D. A. Bureau of Entomology.*)

Miscellaneous Qualities of Plants and Animals.—Aside from yield, every crop has other qualities, of which some are preferable to others. Sugar in melons has been found to depend on several genes; glucose and fructose are in the main dominant, sucrose mostly recessive (Arasimovich 1934). Long fiber in cotton, valued because of the strength it confers on cotton fabrics, is nearly dominant over short fiber in F_1, but there is some variability in length of fiber in F_2 (Fig. 143), indicating that more than one pair of genes is concerned. Yellow color of the flesh of peaches is dominant over the less desirable white. The sugar content of beets is different in different varieties, and it is partly inherited, since there has been some improvement of it through selection. Seedlessness of grapes is inherited, and is probably dominant, for a cross of a slightly seedy variety used as a female

and a seedless variety as pollen parent yielded a seedless variety of commercial promise.

The polled (hornless) condition of cattle, valued as a contribution to safety, is dominant over horns. Color in horses, though not thoroughly understood, is inherited after a multiple-gene plan as in other mammals, with gray dominant or epistatic (page 149) to all others. Knowledge of this color scheme should aid in obtaining matched teams. A high percentage of butterfat increases the value of milk, and there seem to be one or more

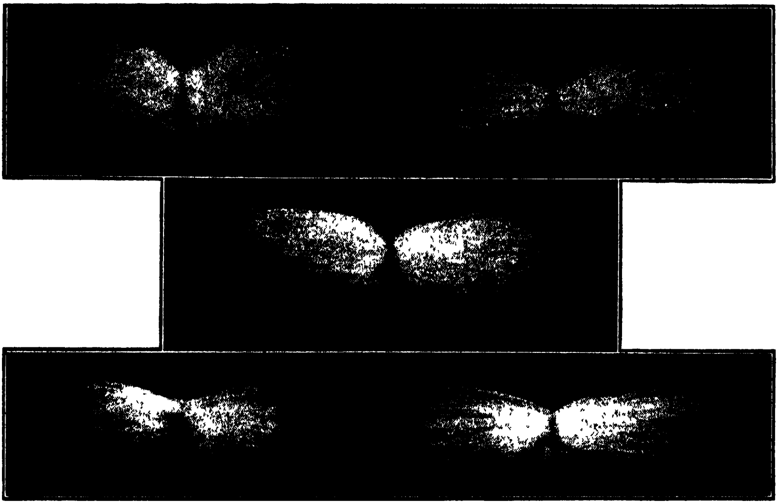


FIG. 143.—Inheritance of fiber length in cotton. Above, at left, short-fibered Holdon cotton; at right, long-fibered Pima. Center, the F_1 generation, with fibers nearly as long as the Pima. Below, extremes of length in F_2 generation. (*Modified from Kearney in Journal of Heredity.*)

genes for this richness apart from those for yield. High butterfat is in general recessive, or nearly so. One variety of bees produces honey rich in levulose, another variety honey with much dextrose. The levulose type appears to be dominant, for the F_1 generation of a cross produces levulose-rich honey, while a backcross of the F_1 to the dextrose type produces intermediate honey, presumably due to a mixture of the two types of workers. Most breeds of animals have association standards which breeders try to meet and which are distinct from productiveness. For the most part the inheritance of these arbitrarily favored characters is not

understood, so that the problem of "improvement" is a continuing one. In skunks the pelt is nearly always marked with greater or smaller patches of white. Since fashion demands a solid black fur, the skins have to be cut and patched. There is, however, a "star" pattern in which the spot is small (Fig. 144), and this variety is being bred. Star pattern is dominant over that with larger white areas. A solid black variety exists as a rarity in nature, and it would seem to be possible to rear this type.

The most spectacular development in fur farming is that relating to the silver fox. The fur of this animal is inherently beautiful, and because of the bands of white on some of the guard hairs it is difficult to imitate. Beginning on Prince Edward Island in 1894, silver fox farming reached boom propor-


FIG. 144.—Fur patterns of skunks. Left, the common variety with large white areas; right, the star pattern. (*From Detlefsen and Holbrook in Journal of Heredity.*)

tions in 1910 and again in 1923, when speculators who had never seen the animals paid thousands of dollars for a single breeding pair. By 1927, however, fox breeders had settled down to raising the animals for their fur. According to current hypothesis, color in the common red fox species is dependent on two pairs of genes. Consequently there may be nine genotypes, mostly regarded as phenotypically different, as follows:

$AABB$ = Alaskan red | $aaBB$ = Alaskan black (silver)
$AaBB$ = cross | $aaBb$ = sub-Alaskan black (silver)
$AABb$ = smoky red | $AAbb$ = standard black (silver)
$AaBb$ = blended cross | $Aabb$ = substandard black

$aabb$ = double black

It would be strange, however, if this were the entire scheme, in view of the many known genes for color and pattern in other mammals.

These few examples illustrate a much longer list of qualities which are not involved in quantity, but which are nevertheless sought among the results of the improvement process.

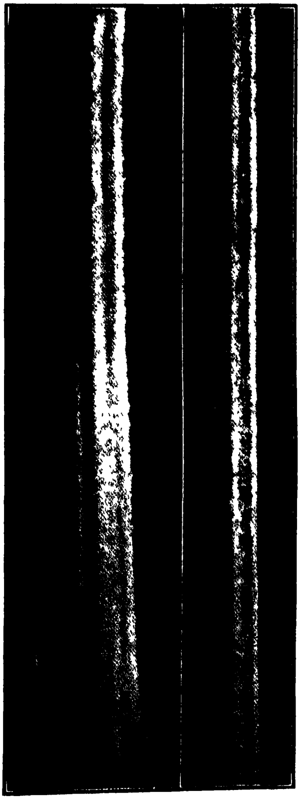


FIG. 145.—Barbed and smooth awns of barley; a seed with awn attached, portion of barbed awn of Manchuria variety, and portion of smooth awn from Lion variety. (*From Hayes in Journal of Heredity.*)

Synthesis of Varieties.—When a number of single desirable characters are known in different strains of a plant or animal species, the task of the breeder is to combine the several preferred qualities in one variety through hybridization. Numerous examples of such synthesis are available in the improvement of wheat and other cereals. In one such project, an Egyptian wheat resistant to one common form of rust and another which was resistant to another form were hybridized to obtain a strain resistant to both. In another campaign of improvement, one variety with a good yield was crossed with another having good bread-baking qualities to obtain a variety with both qualities. To them was added resistance to yellow rust from a third variety, resistance to mildew from a fourth, and resistance to drought from a fifth.

Other qualities desirable in wheat are strong straw, winter hardiness which is dependent on several genes (Worzella 1935), and absence of the beard or awn. The last-named quality makes it easier to handle the unthreshed crop, and is valuable in other cereals besides wheat. In barley Hayes found it undesirable to remove the awns because they were in some way correlated to yield, so he rendered them harmless by removing the barbs from them (Fig. 145) through hybridization.

In tobacco likewise new varieties have been made to order. A good variety must burn rather freely, leave a tenacious ash, and possess a good flavor. The wrapper leaves should have a delicate aroma and a uniform greenish-brown color, be lacking in coarse veins, and be thin, strong, elastic, and broadly rounded at the tip. The tobacco plant should mature quickly, have no lateral branches, produce many leaves per plant, resist disease, and stand up against wind and rain. Broadleaf has three of these qualities not found in Sumatra, and Sumatra has two of

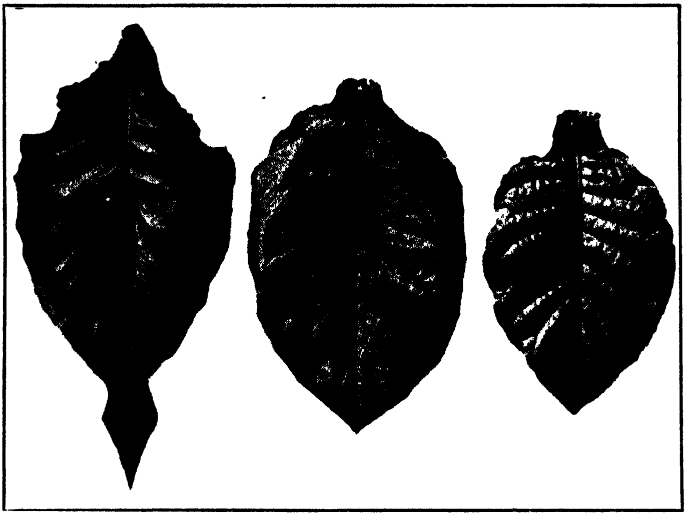


FIG. 146.—Production of a new tobacco variety by hybridization. Left, Broadleaf variety; right, Sumatra; middle, Round Tip, produced by crossing Broadleaf and Sumatra and selecting from their progeny for several generations. (*Reprinted by permission from Jones, Genetics, John Wiley & Sons, Inc.*)

them not present in Broadleaf. Several others of the right characters are in both of them, and in three respects a variety intermediate between them would be better. East and Jones, by hybridizing Broadleaf and Sumatra and selecting for four or more generations, produced Roundtip (Figs. 146 and 147) which is near the ideal.

Egg Laying in Fowls.—Among the remarkable successes of the breeder in improving a quality of economic value, the increase in egg-laying capacity in poultry is singled out for mention apart from any single principle involved. A good egg layer begins producing at an early age (October or November of its first year),

lays steadily with few idle days, is seldom broody, and continues laying through the summer. According to Hurst, who worked with white Wyandottes and Leghorns, early maturity is dominant, rapid fall laying recessive, broodiness recessive, rapid winter laying dominant, and rapid spring production dominant.

The most important single element of production appears to be persistency, or laying late in the fall. A hen that performs well in this respect nearly always receives a high rating in total annual productivity. Hays (1936) finds that high-producing and low-producing hens begin to differentiate at about the 270th day of their first laying year. Those which continue to lay considerably beyond that time are usually good layers, though there is some overlapping. He finds high persistency to be a simple dominant autosomal character.

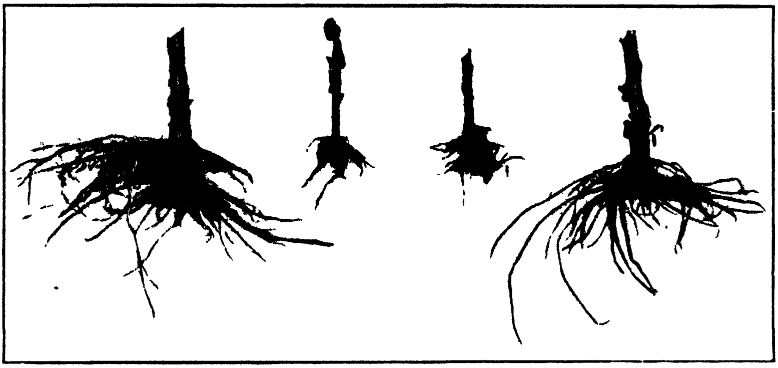


FIG. 147.—Resistance to root rot in tobacco. Roots from an infected field. Roots of the new Round Tip variety at left and right, those of Havana in the middle. The new variety is much more resistant. (*From East and Jones in Journal of Heredity.*)

By selection Goodale raised the annual production of a flock of Rhode Island reds, over a period of 9 years, from 114 to 199 eggs, reduced by 55 days the average age at time of laying the first egg, and increased the average winter production from 28 to 67 eggs. Two-hundred-egg hens are no longer rare, and the best record now is practically one egg a day for the entire year.

Hybrid Vigor.—In certain organisms hybrids—at least some of them—possess a greater vigor than either of their parents. The counterpart of this phenomenon is that inbreeding results in a diminution of vigor. Corn is a particularly good illustration of both effects. Under field conditions the pollen, which is produced

in the tassels at the tops of the plants, falls and is blown by the wind upon the silks of the same plant and of neighboring plants at random. There is some inbreeding, therefore, and a good deal of cross-fertilization, but by human intervention either one may wholly replace the other. Pollen is prevented from escaping by putting paper or cellophane bags over the tassels; and silks on the growing ears may be prevented from receiving pollen by similarly covering them. When the silks are in the right stage for pollination, pollen is sprinkled on them by hand. Pollen from the same plant is used if self-fertilization is desired, but pollen from another plant or another strain if cross-fertilization is to be effected.

If a strain of corn is self-pollinated, a number of generations in succession, it almost always becomes less healthy. Stalks become shorter and slenderer, the leaves have a paler color, and the ears are smaller. Since size of ears largely determines yield, the crop from successively self-pollinated corn becomes less and less.

Now, if two such weakened varieties are crossed, the F_1 plants raised from their hybrid seed are almost always much larger and healthier, as first shown by G. H. Shull and by East and Hayes. The ears of these F_1 plants are considerably larger, and the yield is accordingly increased. The effect on both the plant and the yield is illustrated in Fig. 148. The two plants at the left below belong to two strains weakened by repeated self-pollination, and the corn in the two baskets at the left above is the yield obtained from them on a certain small area of ground. When these strains were crossed, F_1 plants like the third one in the figure were produced, and the yield from the same amount of ground is shown in the third basket above. The yield is about three times that of either parent strain. If, however, the F_1 plants are self-pollinated and their offspring are self-pollinated, and so on, there begin at once a weakening of the plants and a diminution of the yield.

In about six years the beneficial effect of cross-fertilization has completely disappeared; hence it is necessary to repeat the crossing annually in order to gain the full advantage of the increased vigor. On the farm this is done by planting the two strains to be crossed in alternate rows and then clipping off all the tassels of one strain. The ears grown on the clipped plants

will bear the hybrid seeds from which the next year's crop is obtained. Commercial breeders are now doing this crossing on a large scale and selling the F_1 seed to farmers ready to plant. Before doing the crossing, however, it is necessary to decide which two of the many available strains should be crossed. They do not all produce equal improvement on crossing, and only

FIG. 148.—The effect of self-fertilization and cross-fertilization on vigor and yield in corn. (*From Jones in Conn. Agr. Exp. Sta., Bull.* 266.)

by testing them, two at a time can the best combination be discovered. The choice of the two most favorable strains is an expensive operation, and should be done by an experiment station rather than by individual farmers.

Hybrid vigor has been observed in radishes, hemp, rye, wheat, cucumbers, and tomatoes, and in pigs, cattle, and sheep; but no organized attempt has been made to take advantage of it commercially in any of these. The phenomenon is much less well

marked in most of them than it is in corn, and the cost of effecting the crosses would be much greater in some of them.

The prevailing theory of hybrid vigor is that the two strains crossed happen to possess genes which are complementary to one another. Vigor requires certain combinations of genes, and each variety contributes some which the other lacks. The strains "nick," as the practical breeder says. The empirical choice of two strains by the experiment station is simply the discovery of those varieties which thus fit each other. If this is the correct explanation of hybrid vigor, then it should be possible, by sufficiently long hybridization and selection, to get all the necessary genes into one strain, which could be made homozygous for them. Were that done, annual crossing would no longer be necessary. But the labor of synthesizing the homozygous variety could easily be greater than the extra labor of crossing each year, especially since other conditions might sometime change so that the synthesized type would no longer be best.

Legal Applications.—Among the various biological disciplines, pathology, physiological chemistry, and psychiatry have long been of aid in settling legal questions. Only in the last several decades has a knowledge of heredity been similarly used. One type of litigation which such knowledge may aid is that relating to disputed paternity. To help in such cases, the parties to the controversy must differ or resemble in some character whose heredity is understood. One of the first applications of this sort was made in Norway, where by law illegitimate children have the same right to the father's name and property as the legitimate children have. In the case in question a child born out of wedlock had brachydactylous hands and feet, while the mother and all her relatives were normal. The alleged father was brachydactylous, and no other man in the neighborhood had this character. He was accordingly adjudged guilty. Short fingers are too uncommon to figure in many lawsuits, but its very rarity enabled the court in this instance to fix the guilt upon a single individual. Had the character been a common abnormality, all that the court could have decided from the facts of heredity would have been that the father was some brachydactylous person.

To be of frequent use in the courts, the inherited characters involved must be those in which many people differ from many

others. Eye color would be excellent if it were a little less erratic in its expression. At the present time one of the most reliable characters is the blood group. Everyone belongs to one or another of four blood groups, the mode of inheritance is known (page 110), and the technique of determining the group is subject to few errors. If the blood group of a child and that of one of its parents are known, the blood of the other parent is limited to certain groups, as in the table below.

Blood group of child	Blood group of known parent	Blood group of unknown parent
O	O	O, A, or B
O	A	O, A, or B
O	B	O, A, or B
A	O	A or AB
A	A	O, A, B, or AB
A	B	A or AB
A	AB	O, A, B, or AB
B	O	B or AB
B	A	B or AB
B	B	O, A, B, or AB
B	AB	O, A, B, or AB
AB	A	B or AB
AB	B	A or AB
AB	AB	A, B, or AB

Although in a few instances an unknown father might belong to any of the groups, in some combinations of child and mother he would be limited to three of them and in a few of them to two of the groups (see last column of table). No man could in this way be proved to be the father of a given child without the aid of other evidence, but he could often be shown not to be the father. Thus, a man of blood group AB could not be the father of a child of blood group O no matter who the mother was (first three lines of the table), and a man of group A could not be the father of a child of group B if the mother were of group O (eighth line).

Sometimes the problem is to decide which of two children might belong to a certain pair of parents. Instances of this sort have arisen in hospitals where babies could be inadvertently

exchanged. A case of such exchange in a metropolitan hospital a few years ago was decided beyond doubt by determination of the blood groups. A similar question arises when a possible impostor claims to be the long-lost child of wealthy or titled parents, and the same possibility of an answer from the blood groups exists. In both of these situations the parents are known and the child is uncertain; or the child is known and both parents are uncertain. The possibilities open to the unknown child are shown in the following table.

Parents	Possible Children
O × O	O
O × A	O, A
O × B	O, B
O × AB	A, B
A × A	O, A
A × B	O, A, B, AB
A × AB	A, B, AB
B × B	O, B
B × AB	A, B, AB
AB × AB	A, B, AB

Possibilities of legal tangles that call for knowledge of heredity were created when the Congress, in May, 1930, extended the federal patent law to cover new plant productions. To be eligible for patent, the variety must be capable of propagation by some vegetative method, as by cuttings, grafts, or bulbs. Tubers (as of potatoes) are, however, excluded. The requirement of vegetative reproduction is made in recognition of the fact that seed, due to their origin from two parents, are notoriously variable on account of recombination. Several hundred varieties have been patented under this law. So far one test has been made in court (Barrons 1936). The court was unable to say, from the characteristics of the alleged infringing plant, that it *must* have come from the patented stock. In another suit the patent was declared invalid because the variety was "introduced" before passage of the law.

Eugenics.—The application of knowledge of heredity to improvement of the human race, referred to in the opening paragraph of this chapter, constitutes the science of eugenics. Because of its complicated nature, and because natural human interest necessitates a more extensive treatment, this subject is segregated in a separate chapter.

CHAPTER XXVII

EUGENICS

Plans designed to improve the human race, or, more correctly stated, to breed it toward a type, are not at all new. The proposal now occasionally made that children be regarded as assets of the state alone, and as such be put under state control, is at least as old as Plato, who, in his "Republic," outlined a scheme for developing an ideal governing class, which apparently was the only group that greatly concerned him. Plato proposed that matings were to be made by the magistrates, from among superior individuals, while the children of inferiors, if they could not be forestalled, were to be destroyed. Plato's plan was not wholly ingenuous; for, recognizing that the decisions of the magistrates would cause trouble, he suggested that matings should be determined by lot, which, however, was to be manipulated in some way so as to give an appearance of being fortuitous.

A scheme almost as arbitrary as that of Plato was actually employed in Sparta, whose population was essentially military. Marriage was permitted in order to supply new soldiers. Children were preserved or not, according to the decision of the rulers. The scheme worked well for a time, if by this is meant that it succeeded in doing what it was designed to do, namely, breed a race of soldiers. Sparta is known to the present time chiefly for its severity and its military superiority; it had nothing of the art, science, or literature so characteristic of Athens. When, by intercourse with the rest of the world, Sparta discovered something else than combat to live for, the old stern regime gradually disappeared. Although, since then, many tribes and nations have destroyed their unfit, the purpose was probably one of consideration for their own convenience rather than from any thought of the next generation; and, from the time of Sparta to within the last few decades, one looks in vain for well-defined movements aimed at human improvement through heredity.

Sir Francis Galton.—The name and the early direction of the science of human racial improvement are to be credited to Sir Francis Galton (Fig. 149), who had for years devoted himself to a study of heredity in man and had conceived the idea of applying the newly acquired knowledge for man's own benefit. In his book "Inquiry into Human Faculty and Its Development," published in 1883, he writes:

FIG. 149.—Francis Galton. (*From Galton, Memories of My Life, Methuen and Co.*)

We greatly need a brief word to express the science of improving stock, which . . . especially in the case of man takes cognizance of all influences that tend . . . to give the more suitable races or strains of blood a better chance of prevailing speedily over the less suitable. . . . The word *eugenics* would sufficiently express the idea.

Galton's words are repeated because they clearly indicate that he had in mind improvement of average human qualities through heredity. This is the significance attached to the word by pure biologists. Unfortunately, in popular usage much has been included under the term which, while it concerns improvement, has nothing to do with heredity or racial values. Most of this extraneous matter is hygiene. In the discussion of eugenics in this book, the early definition of the term is adopted.

The Eugenics Problem.—The need of racial betterment may be clearly visualized through a glance at the extremes of social worth. At the bottom of the scale, one finds in Great Britain and Ireland 178,000 mental defectives (1921); or, according to the estimate of R. A. Fisher, 1 in every 16 in a group making up 5 per cent of the population. Other writers regard this estimate as much too low. The best estimates of the frequency of feeble-mindedness for various regions, most of them in the United States, range from 1 in 294 to 1 in 138. In Indiana, 2.1 per cent of the population of ten counties is said to be mentally deficient. If the rate is the same through the rest of the state, this means a total of 56,000. More than half of these require institutional care. Indiana has maintained a registry since 1889, and on it are the names of 158,000 who are now or were in the past state charges. That the number is not decreasing is indicated by cases like the following, cited by Butler. One feeble-minded woman had 11 illegitimate children; one of her daughters, also feeble-minded, had 8 illegitimate children, 7 of them feeble-minded; and one of these 7 has had 4 illegitimate children. All told, the first of these 3 women has 56 direct descendants, 31 of them feeble-minded, 18 of them having been in institutions, and 16 of these having cost the state $10,800 in support. Four generations of the feeble-minded have been found in one institution at the same time, all living at state expense. In the United States as a whole, according to an estimate by the White House Conference in 1930, 2 per cent of the population is definitely feeble-minded. It is estimated there are 6,000,000 in the United States who have been, are now, or will sometime be legally committed as insane to state institutions. If epilepsy is as prevalent everywhere as in the states where they have been carefully estimated, there are 150,000 of its victims, only part of them in institutions but in general constituting an economic burden. One might add to these the paupers and petty criminals, though many, perhaps most, of these would fall also in one of the previously mentioned classes.

At the opposite end of the scale of native ability is a group of men and women who cannot be counted because reliable criteria of recognition are wanting and who cannot be priced because there is no unit of value. They are the leaders in every field—' art, science, literature, religion, government, invention, educa-

tion, business. The dependence of humanity upon a relatively small number of these people will probably never be generally appreciated. Practically all our advancement in intellectual, artistic, moral, civic, and commercial lines is derived from them. It is not necessary to magnify their individual significance, as has sometimes been done. Thus, it has been said that if several hundred leading individuals in New York City were suddenly abducted, the metropolis would be hopelessly crippled. So it might for a time, but there are doubtless some hundreds of others who could step into their places and fill them acceptably if given the opportunity. It is not so important that the actual leaders of any given moment be preserved, as that the class from which leaders may be recruited be maintained.

Between these extremes is the bulk of the general population, ranging in value from the near-defective to the near-great. It is the problem of eugenics to abolish or reduce one of the extremes of social worth and to increase or at least maintain the other. There is a difference of opinion as to which of these steps is the more urgent, some maintaining that the burden created by the defectives must first be lifted, others holding that if the superior class can be sufficiently supported, progress will be insured notwithstanding the drag of the subnormal. These tasks are not equally easy. They cannot be left to perform themselves. Under the present practices of human society, it can scarcely be doubted that the race is deteriorating. The mode of life of earlier ages tended to destroy the unfit and increase the superior. Customs deliberately chosen frequently insured the former, and every opportunity favored the latter. Today we nurture the unfit, while it is a question whether the superior are maintaining their numbers.

The Eugenics Program.—What is to be done to better the situation just described? The answer to this question constitutes the eugenics program. While there is no "official" program, because there is no single group or organization charged with formulating one, there is general agreement that work in eugenics may properly be of four different kinds, research, education, conservative legislation, and administration.

The first of these, research, is fundamental to the others and at present the most urgent. The need of more accurate knowledge of the mode of inheritance of mental characteristics and of

means of recognizing mental mutations is stressed. The investigation might well include marriage selection; differential fecundity; the effects of war, immigration, and hybridization of races; and birth control with its actual and potential consequences. It is well established that the birth rate is higher in rural districts than in cities, and that there is a drift of the population from country to city; and it is desirable to know whether removal to the city is the cause of the reduction of the birth rate, or whether those who move to the city are merely those who would have smaller families anyway. Is the influence of college education uneugenic? Does accumulation or inheritance of wealth tend toward sterility? Are modern medicine, sanitation, philanthropy, and religion so conducted as to serve eugenic ends? These are questions whose answer the proposed research program would seek.

The education involved is partly formal, partly popular. The formal education is designed to reach the teachers and other agencies for accurate dissemination of knowledge and includes courses in eugenics (always with their foundation in principles of heredity) and in medical and other professional schools. Physicians have, as yet, too strongly the environmental point of view in relation to disease, and it is thought that lawyers, judges, ministers, diplomats, and consuls would be greatly aided by knowledge of eugenics. Popular education is to be effected through the press, religious organizations, lecture associations, extension bureaus, congresses, radio addresses, and so on. Information so diffused must be accurate, not premature or exaggerated, and should be given with earnestness and seriousness. The purpose of this educational program is partly for the voluntary use of individuals receiving the instruction, partly to prepare a public opinion upon which the remainder of the program may securely rest.

The legislative part of the program should develop slowly, or even wait for a time. Among the laws regarded by some, or many, eugenists as ultimately desirable are those providing segregation of criminals and other defectives, suitable regulation of marriage and divorce on biological principles, limitation of immigration on the basis of native ability, differential salaries of parents dependent on size and tested mental qualities of families, differential taxation (presumably income and inheritance taxes, in

particular, on basis of size and tested qualities of families), legalization of proper birth-control information, establishment of eugenics bureaus, and registration of family pedigrees. Prevention of child marriages should keep some inferior families from getting an early start, ahead of their superiors. The list of desirable enactments may well be allowed to remain in a fluid state, because in it are many recommendations concerning which there are grave differences of opinion, and, in the meantime, education of the public to support some legislation and further determination of the facts upon which laws will have to rest should proceed. Part of this legislative program is discussed further below.

The administrative reforms desired relate to standardization of records in institutions so that they will be comparable, preservation of more complete and more biological records by courts and custodial institutions, and accumulation of more extensive and better vital statistics.

Execution of the Program.—Part of the eugenics program is already under way. The research is being carried on by such organizations as the Eugenics Record Office (part of the Department of Genetics of the Carnegie Institution of Washington), the Eugenics Research Association, various state and federal bureaus, hospitals for defectives, municipal courts, and individuals in universities and other institutions. Much is being done, but there is room for many times as much more. Since the remainder of the program must largely await the results of research, these investigations cannot be begun too soon or pressed too rapidly to a conclusion. Special mention should be made of the part which individuals may play in making known their own family histories or any others they may know or be able to discover with sufficient accuracy. Families which can be traced for several generations, in which numerous individuals including the collateral lines are known, and in which fairly accurate descriptions or estimates are available, are much desired. Anyone able to supply such information should communicate with the Eugenics Record Office, Cold Spring Harbor, Long Island, from which instructions relating to procedure will be sent. Information furnished to such institutions is always held in the strictest confidence.

The educational part of the eugenics movement will, in all probability, keep pace with the research and will be stimulated

by it. This is particularly true of the formal education proposed. Scientific courses in colleges and universities are generally designed to be informative. Good advice, discussions of ethics, and matters of sentiment may be, and usually are, included when they are at all relevant; but seldom can such instruction be made successful unless the emotional appeal is fairly balanced by a body of ascertained fact. When this body of fact is enlarged, the appeal may be expected to expand and its influence to increase. Even the popular educative movement should yield more results with the growth of the actual knowledge whose application it seeks to promote.

On the legislative side, almost all that has been done relates to the elimination of certain defectives. Although there are laws on marriage, divorce, illegitimacy, mother's pensions, child labor, etc., most of these enactments do not have a eugenic purpose or are ill designed to serve that purpose. With respect to defectives, it seems wise to limit present legislation to those traits whose heredity is best known. Possibly only hereditary feeble-mindedness and perhaps epilepsy should be subject to legislation at present. There are already many state laws which aim to prevent reproduction by defectives. Most of them have been made too inclusive, often being aimed at criminality and sometimes at tuberculosis and other diseases. This has no reference to venereal diseases, which it is proper to curb in any practical way.

If legislative attempts to control reproduction of the unfit could be restricted to hereditary feeble-mindedness and allied mental defects, the procedure could follow either of two courses. Segregation of defective men and women in separate institutions appeals most to some eugenists. It has two drawbacks; it is expensive, and the defective individuals are not always kept in the institution. The chief alternative method is sterilization, by means of an operation which makes reproduction impossible. In the male, this operation is comparatively simple and causes little inconvenience; in the female, it requires a stay of two or three weeks in the hospital.

Progress of Sterilization.—The first sterilization operation for eugenic reasons was done by a German surgeon in 1897 (Popenoe 1934). Official sterilization in the United States began, with the patient's consent, in 1899. Indiana was the first state to adopt a law providing for compulsory sterilization under

certain conditions. That was in the year 1907. A few other states followed within a year or two, and up to the present time 32 states have or have had sterilization statutes. In New York, New Jersey, and Nevada the laws were declared unconstitutional and were not re-enacted in modified form. In several other states the first laws, and sometimes the second, were declared invalid, but bills designed to avoid the unconstitutional feature were later passed. The features that were most commonly the causes of nullification were (1) application only to inmates of institutions, which made the laws class legislation, and (2) inclusion of an element of punishment in them. When the laws came to be wholly corrective, not punitive, and to rest on a strictly biological basis and applicable to everyone who met the proscribed conditions, courts generally held them valid. The United States Supreme Court ruled in favor of the Virginia law in 1927, and it seems entirely possible now to draw the statutes in such form that they will be constitutionally valid. Twenty-nine states now have such laws in force. In most of them they are compulsory, some provide both voluntary and compulsory procedure, and one (Vermont) allows only voluntary sterilization.

In some states the law has seldom been used. In others there have been thousands of operations. The extent to which sterilization has been practiced in state institutions under law is indicated by the table on page 313, which does not include operations done with the patient's consent for purely therapeutic, not eugenic, reasons.

This table shows that California is by far the most active state in pressing this particular eugenic reform. Operations are continually increasing, the number for the year 1936 having been 2,241 for the United States as a whole.

In other countries the most ambitious scheme of sterilization is found in Germany, where the operation was decreed for possessors of a wide variety of characters. To govern the procedure 205 eugenics courts and 26 superior eugenics courts were created. In the first year of operation these courts passed upon 64,499 cases, about three-fourths of those before them, and ordered 56,244 sterilizations (Cook 1935). The provisions of the law are given by Popenoe (1934). Sweden and Norway now have sterilization laws, Denmark passed one in 1929, one of the Swiss cantons a little earlier, the state of Vera Cruz in Mexico in 1932,

EUGENIC STERILIZATIONS IN STATE INSTITUTIONS, TO JANUARY 1, 1937

State	Female	Male	Total
Alabama	95	129	224
Arizona	10	10	20
California	5,551	5,933	11,484
Connecticut	372	23	395
Delaware	231	263	494
Georgia*	0	0	0
Idaho	10	4	14
Indiana	228	321	549
Iowa	46	61	107
Kansas	711	1,039	1,750
Maine	115	14	129
Michigan	1,315	381	1,696
Minnesota	1,054	224	1,258
Mississippi	223	99	322
Montana	62	34	96
Nebraska	189	123	312
New Hampshire	281	45	326
New York	41	1	42
North Carolina	325	65	390
North Dakota	250	84	334
Oklahoma	113	42	155
Oregon	727	378	1,105
South Carolina	0	0	0
South Dakota	194	110	304
Utah	60	46	106
Vermont	98	41	139
Virginia	1,557	1,077	2,634
Washington	165	31	196
West Virginia	10	0	10
Wisconsin	696	96	792
Totals	14,729	10,674	25,403

* Georgia law passed in 1937.

and two Canadian provinces (Alberta, British Columbia) in 1928 and 1933, respectively.

The available data concerning sterilization are mostly those from institutions which perform the operation with the sanction or at the mandate of state law. Since a patient may be unofficially sterilized at his own request by private physicians, who do not report such cases, the actual number of operations is doubtless greater than here indicated.

It is difficult to make recommendations regarding the extension, control, or guidance of the practice of sterilization. It would work much better if the exact genetic basis of all human defects could be known and particularly better if heterozygous individuals could be detected. A committee of the American Neurological Association (Myerson *et al.* 1936), in a book which is generally critical of the conclusions of most students of human heredity, recommends that sterilization be considered only for people suffering from Huntington's chorea, hereditary optic atrophy, Friedrich's ataxia, hereditary feeble-mindedness, dementia praecox, manic-depressive psychosis, and epilepsy and that by no means all of these be actually sterilized. The attitude of this committee may be otherwise judged from its further recommendation that sterilization be voluntary and regulatory rather than compulsory.

Is Elimination of Defectives Possible?—Some concern has been shown by various writers as to whether by either of the methods outlined, segregation or sterilization, it would be possible to eliminate the defectives in our midst or even to reduce them seriously. Feeble-mindedness is rated as an approximate recessive, and as such should be carried along concealed in individuals who might be suspected as carriers but against whom neither plan of elimination could operate. A rather discouraging computation indicated that starting with a population in which there is one feeble-minded person in a hundred, and assuming random mating, it would require 22 generations to reduce the number of defectives to 1 in 1000, and 68 generations more (or 90 in all) to reduce it to 1 in 10,000. If this decrease were spread evenly, by percentages, over the entire 90 generations, the process might seem hopelessly slow, and not worth the energy devoted to it. R. A. Fisher has pointed out, however, that the very first generation of rigid elimination reduces the defectives by more than 17 per cent, that two generations of elimination reduce them by 30 per cent and that three generations diminish them by over 40 per cent. This is all on the assumption that at the beginning there was 1 per cent of defectives in the population. If the defectives were less numerous at the beginning, the progress would be somewhat slower. Fisher also shows that, because there is much inbreeding among the defectives, instead of random mating among all classes, the rate of reduction is greater

than that just calculated. If, in addition, feeble-mindedness is incompletely dominant so that heterozygotes may sometimes be recognized, the reduction should be still more speedy. The effect of such elimination is not, therefore, so slow as wholly to discourage the attempt. This computation does not, of course, take into account the types of feeble-mindedness that are not inherited.

Increasing the Superior.—Reduction in the number of defectives, which is contemplated in most of the measures so far discussed and which may be called the negative part of the eugenics program, does not add anything to the higher levels of human value. Though the average is raised by eliminating the worst, the best is no better than it was before. What is more serious is that the best hundred thousand are not any better because the lowest hundred thousand have been removed. It is the generally superior class, rather than a small group of geniuses, which is the concern of positive eugenics.

Many measures have been proposed for preserving and if possible increasing this superior class. The first problem is to recognize it, for superior people often spring from parents who by ordinary standards were not in any way remarkable, and mediocre offspring often come from very gifted parents. Pearl points out that very few of the people who attained such eminence as to receive a page or more of biography in the Encyclopaedia Britannica had parents who were in any way distinguished. He states that if during the past 2,000 or 3,000 years breeding had been restricted to those who were eminent, 95 per cent of the great people who existed in that time would never have been born. Such criticisms merely mean that we have not yet discovered the really applicable criteria of superiority. So long as human traits of different values are inherited, and people differ from one another in their genotypes, there are bound to be superior and inferior classes. The eugenist's first problem is to recognize them.

When superiority can be detected without too many errors, how shall it be increased? Deliberate control of matings by constituted authority is not possible in a democratic society, though it was once attempted through a voluntary renunciation of individual rights. This attempt was made in the Oneida Community, which was founded in Vermont in 1841 and moved to

New York in 1848. The control referred to extended from 1868 to 1879. During this period a group of young men and young women placed themselves at the disposal of the founder of the community, to arrange whatever marriages he deemed desirable. The control was exercised part of the time by the leaders of the community, part of the time by a special committee. Couples desiring to marry made application to this central authority, which rejected about 17 per cent of the applications. Other marriages (about one-fourth of the whole number) were arranged on the initiative of the committee. To these controlled unions there were born 58 children. They showed a better than average longevity; only two of them had any mental deficiency, and these could have been attributed to injury. On the whole, their qualities indicated that the control had not been badly administered.

Differential Birth Rate.—In other communities the choice of mates must usually be voluntary, and the problem of increasing the superior classes resolves itself into a question of proper birth rates. Numbers of children born must be balanced against the death rate to get a net factor for increase, and the secular value of the birth rate as commonly expressed is sometimes altered by changes in the age distribution of the general population. These intricacies are here laid aside, and only general consequences stated. A century ago the economically successful types, which may be roughly identified with the superior classes, had the larger families. There was little limitation of families then. The landed gentry of Great Britain, for example, then had families of 7.1 children. Fifty years later, however, the average number had dropped to 3.1 per fertile marriage. All other available statistics show similar comparisons—a high birth rate for the successful classes, which fell over a period of decades to a moderate or low rate. If all birth rates had fallen proportionately the decline would affect only the population problem (see Chap. XXVIII), not the eugenic situation. But the birth rate of the inferior classes remained high. Later a decline set in for the lower classes also, but so far it has not equaled that for the upper groups. Before this belated decline had proceeded far, families with five or more children at completion, based on a section of the 1910 census, made up 10.9 per cent of the professional classes, 12.4 per cent of the

business group, 21.5 per cent of skilled workers, and 33.6 per cent of the unskilled. In the rural population, whose birth rate is regularly higher than that of cities, families of five or more children were found among 35.2 per cent of farm owners, 49.2 per cent of renters, and 50.2 per cent of farm laborers. To whatever extent these economic distinctions represent native worth, the differential birth rate existing among them is eugenically unfavorable.

In the last two decades, particularly since the World War, the birth rate in some European countries has declined notably in the proletarian classes. This is obvious in the extent to which the total rate has dropped, even when classified statistics are not available. The poorer sections of Stockholm and Oslo in 1928 showed a birth rate only slightly higher than that in the rich districts. In Berlin there is now little difference between the industrial and the well-to-do areas. Information concerning cities in the United States is not generally available, but there has doubtless been a smaller change in the same direction. Most writers attribute the decline to a filtering down of the practice of family limitation from the superior classes, where it has long been prevalent, to the less intelligent groups.

There are minor reversals of the contrast between economic classes. Yale graduates of several classes graduating in the nineties were graded by their colleagues as to their value to humanity, and it was found that those rated high had on the average larger families than those rated low. Similar results were obtained from a study of Harvard graduates, and for those included in "Who's Who in America." Certain colleges can even boast a birth rate among their graduates that is about as high as that of the population as a whole, or a rate among their superior graduates which is higher than that of those who barely passed. While these differences are favorable ones, they do not count heavily in the total birth rate, for all of them belong to a class which is not maintaining itself. They are favorable items in a much larger unfavorable situation.

Net Reproductive Rate.—Offsetting or abetting the changes that have taken place in the birth rate of different classes are changes in the death rate. The total death rate has declined to such an extent in the United States that in the 30 years from 1900 to 1930 eleven years was added to the average expectation of life. This expectation was in 1930 for white females 62.62

years, for white males 59.0 years. This extension of life, if it affected the different classes unequally, as it has done, might upset all calculations based on relative birth rates alone. No startling modification of the conclusions is necessary, however, when birth rates and death rates are balanced to obtain what may be called the net reproductive rate. For professional classes this net rate in the United States in 1928, as computed by Lorimer and Osborn (1934), was 0.76, for business and clerical 0.85, for skilled labor 1.06, for semiskilled labor 1.03, for unskilled labor 1.17, and for agriculture 1.32. These indices mean that the first two classes are not maintaining themselves and will have to be recruited from the others. The rural reproductive rate is doubtless governed in part by factors residing in the mode of life and is characteristically high. It is the low rate of the professional, business, and clerical groups which constitutes one of the major problems of eugenics.

Proposed Methods of Positive Eugenics.—To secure larger families from the classes with greater native ability, several concrete proposals have been made. It must be recognized that the more intelligent groups can never be induced to compete for numbers of children with the lower classes. If some increase in their families can be effected, however, that is a worth-while gain. Education is first of all necessary, because in the present state of popular information on the subject nothing more effective can be made to succeed. Education as to the needs of the race might in rare families lead to voluntary effort to raise the birth rate; but the chief hope from education is that it will lead to democratic support for other steps.

One suggested other step originated with Francis Galton, who proposed that promising young couples be provided good houses at low rentals. An elaboration of this plan is being tried, with the aid of philanthropy, near Strasbourg in Alsace. Qualified young couples are provided furnished houses at low rental, and, if after a reasonable time they have no children, they are required to vacate in favor of others. The birth rate in the community is 128 per thousand married women per year, as against 90 for the general population. If the couples are chosen correctly, the effect can scarcely be other than beneficial; and there is evidence that physically, at least, the children produced are distinctly above the average.

Another proposal is that the income of a family meeting certain requirements be increased at the birth of each child. To avoid prejudicial treatment of capable, married, and prolific parents during enforced layoffs, the increase in wages should not be borne by the individual employer; and to avoid political difficulties, it should probably not come from the state. If such increases were borne by the industry as a whole, temptation to dismiss would be removed. The general effect of such a plan would probably be to lower the wages of single and childless workers, so that the total cost to the industry would be about the same. Missionaries of some churches receive such a family allowance, and their families are considerably larger than those of other ministers of the same denominations.

A system of loans to young couples (canceled in part on the birth of children) has since 1933 increased both marriages and births in Germany. It is doubtful, however, whether the innate qualities of the population will be improved by this method, for the purpose is possibly to increase military strength, and loans may be granted indiscriminately. The plan is of interest as proving that the birth rate can be raised by relieving parents of some of the financial burden of rearing children. If applied only to superior or at least to very satisfactory couples, it should have a eugenic effect. Whether a scheme of family allowances could be adopted in a democratic country is questionable. It would require the enlightened self-interested support of the general population—which harks back to the educational part of the positive eugenics program.

The outlook for putting any of these plans into effect is not bright. Holmes (1936) in a thoughtful and conservative appraisal of the prospects of positive eugenics points out that people are not now interested in improving the hereditary qualities of the human breed. They know little about heredity and have little appreciation of the importance of hereditary distinctions among men. To remove this ignorance and improve the understanding of what is at stake is the first task.

CHAPTER XXVIII

THE POPULATION PROBLEM

While from a eugenic standpoint it is the quality of the human race which is of prime importance, there are many respects in which its mere numbers are of grave concern. The problems raised by growth, decline, or changing age distribution of a population are not only biological but, to an even greater extent, economic, political, and social. The population problem has been in a sense before the public ever since Malthus published his "Essay on Population" in 1798; but very few people, even among the publicists, regarded it seriously until comparatively recent years.

Reversal of Attitude.—In the short time that population has been a subject of frequent and serious study, the attitude of scholars toward the outlook for the future has done a remarkable somersault. From a fear, only a few years old, that there soon would be little room and less comfort for human beings on the earth, the average statistical mind has turned to gloomy forebodings of the time when there may not be enough people to occupy the land most advantageously. Perhaps it cannot be said that any one scholar has reversed his position on this question, but the collective viewpoint of the active students has experienced a profound change.

The contribution of Malthus was to call attention to the rapid rate of reproduction of the human race and the negligible expansion of the habitable portion of the earth and of the means of sustenance. Few people like gloomy predictions, and Malthus was roundly denounced and abused. So long as population continued to grow and to promise future boundless growth, almost no one could be induced to think of the necessity of remedies. In the nineteenth century Europe more than doubled its population, England and Wales more than trebled, and the United States increased over twelvefold. Though these changes were precisely what the Malthusian doctrine predicted and though they must bring these lands nearer to their ultimate

limit of growth, the fact that a limit had not yet been reached led statesmen and others to regard the growth as a refutation of Malthusianism. One of the reasons for the great increase in numbers in that century was the advance of medicine and sanitation, resulting in a greatly reduced death rate. And one of the reasons for the failure of this rapid growth to bring the population sensibly near the limit at which discomfort would begin was the industrial revolution. Manufacturing opened up many new occupations besides agriculture, the mechanization of

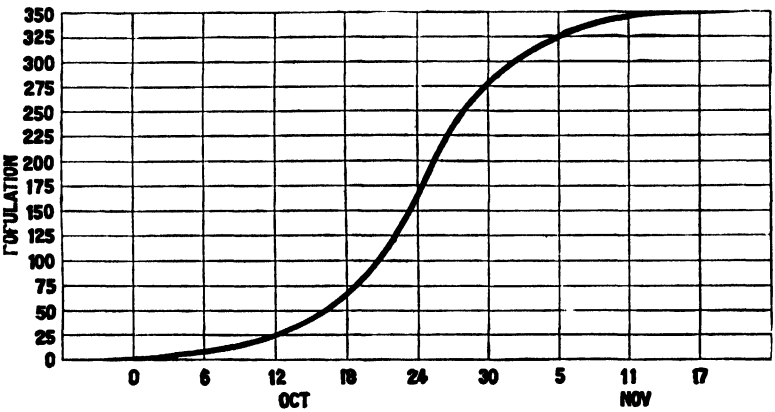


FIG. 150.—Increase in population of vinegar flies in a "universe" of limited size. A few flies were placed in a pint milk bottle with suitable food and allowed to reproduce. A census was taken every few days. The curve shows that the population increased slowly at first, rapidly afterwards, more slowly again, and finally became stationary. (*Modified from Pearl.*)

farming released unneeded labor to go into industry, and the march to the cities began. Furthermore, the substitution of machinery for horses in farm operations has released a great deal of acreage from human food; for it took more land to keep a horse than to feed a member of the family.

Notwithstanding these changes which made the United States, for example, capable of holding in comfort a much larger number than would be possible on farms alone and despite the public apathy to any warnings that Malthus was still right, only premature, most statisticians and philosophers who studied the outlook continued to foretell a time when the standard of living would be lowered because of the growth of population beyond the means of support.

Pearl, experimenting with Drosophila in a bottle, taking a census of the flies at frequent intervals, found that their population grew as indicated by the curve in Fig. 150. They increased more and more rapidly at first, then more and more slowly, until they reached a maximum population at which they remained stationary. When he examined the population of countries in which censuses had been taken over long periods, he discovered that their curves of population growth resembled parts or all of the Drosophila curve. The graph for Algeria (Fig. 151) almost duplicated the entire curve; that for the United States

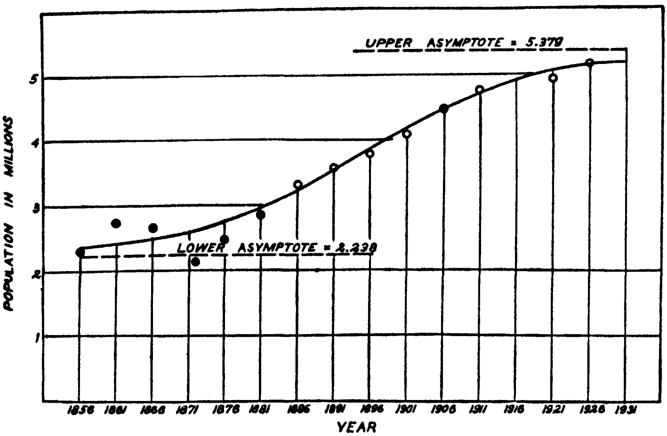


FIG. 151.—Growth of the native population of Algeria over a period of 75 years. (*From Pearl in Human Biology and Racial Welfare, Paul B. Hoeber, Inc.*)

closely resembled the lower half or a little more (Fig. 152); that for France (Fig. 153) was like the upper third. On the assumption that the curve for the United States would continue as did that for Drosophila, Pearl was led to predict an ultimate population of about 197,000,000 reached in the year 2100, with only slightly less than that number in the year 2000. When the 1930 census was taken, Pearl added the necessary extension to the known curve, and found that it fitted reasonably well.

East, assuming that the food supply would be the factor limiting population, examined the probability of increase in tillable lands, of improved varieties of plants and animals, of better methods of cultivation, and of manufacturing food without agriculture. He calculated that with all the most favorable

assumptions the population of the United States might reach 320,000,000 but that Pearl's approximately 200,000,000 was a more likely limit.

Other writers arrived at various figures. Mostly, however, the numbers were higher than that of Pearl; and mostly these writers recognized that the standard of living would ultimately be lower than at present. Mankind could take heed and prevent this discomfort, if it would, by deliberately checking population growth.

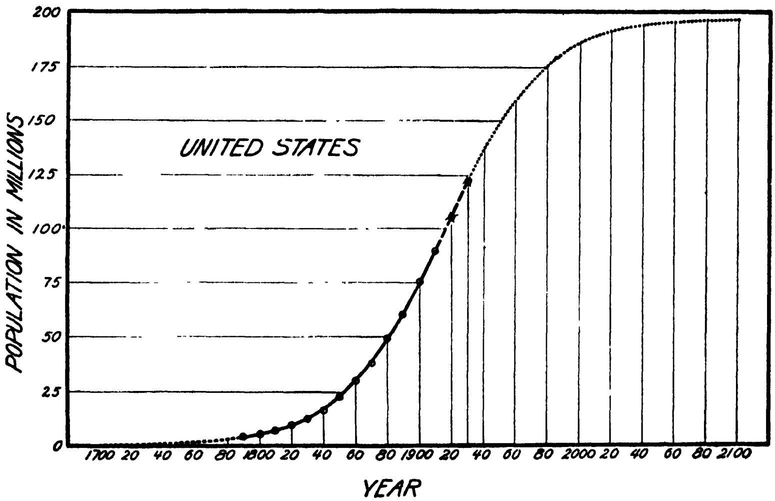


FIG. 152.—Curve of population of the United States, with its projection into the future. (*From Pearl and Reed in Science.*)

Not all statisticians were convinced of the correctness of these conclusions. Moreover, there has been an important change in the rate of increase of mankind at many places on the earth. Taking into account this reduction in the net growth of population, practically all recent demographic studies have led to the conclusion that for the United States and a number of other countries the maximum population will be reached much earlier and that there will be far fewer people than had been predicted. This reversal of opinion came almost suddenly, and the possible consequences are contemplated with considerable misgiving. Instead of dreading a prospective shrinkage of the family diet when there would not be food enough to go round, food producers are now wondering who will buy their products. Everybody

with something to sell will see the growth of markets decline or even be reversed. Less of everything will be needed than we have heretofore anticipated. Early in the new order there should be particularly fewer young people. Schools will enroll fewer pupils, school buildings be only partially filled, teachers be in oversupply wherever they have been adequate heretofore. Manufacturers of baby carriages, children's clothes and toys will necessarily turn to other products. These are some unpleasant predictions coming out of nearly every demographic conference, congress, or federal department study, in recent years.

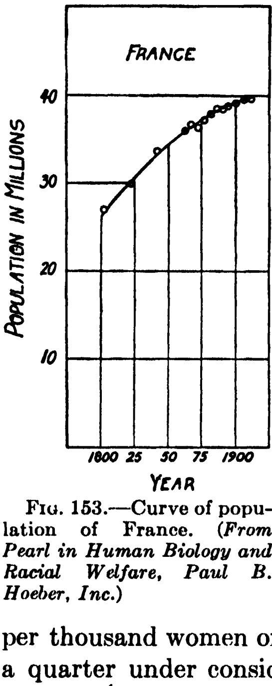


FIG. 153.—Curve of population of France. (*From Pearl in Human Biology and Racial Welfare, Paul B. Hoeber, Inc.*)

Birth Rate and Age Distribution.—The occasion of these gloomy prophecies is the fall of the birth rate in a number of countries. Before the Civil War in the United States the population doubled every 25 years, and the center of population moved steadily westward. After the Civil War the increase was no longer geometric, but amounted to roughly 15,000,000 every decade. Something of the nature of this change can be seen in the number of children per thousand women of child-bearing age during the century and a quarter under consideration (Fig. 154). Since 1930 the population has not even maintained itself. The change began in the early or middle twenties. In 1921 about 3,000,000 children were born annually in this country; in 1924 about 2,900,000; and since 1933 about 2,300,000.

Even at this rate the casual observer would say that the population is increasing satisfactorily, for the death rate is low and there is a net increase of about 800,000 a year (O. E. Baker). This increase is, however, illusory because of the age distribution of the population. At the present time the United States is a nation of the young and middle aged, with relatively few old people. Deaths are therefore relatively few. This mild preponderance of youth is due to a high birth rate and immigration *in the past.* With the passage of time the middle-aged will

become old and deaths will increase. On the basis of present rates, deaths may then be expected to exceed births, and the population decline except as immigration adds to it. Hence, while the population is now increasing, the same birth rate will in a few years not be sufficient to maintain it.

To illustrate the effect of age distribution on the indices of population growth, an extreme example will be useful. It is, of course, hypothetical. Suppose that a population consists only of couples between the ages of 30 and 40, who are normally fertile but at present without children. In the next few years the birth rate in this population will be high wnen expressed, as usual, in

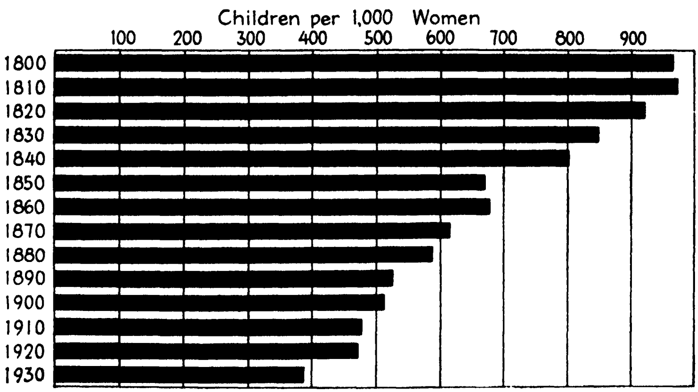


FIG. 154.—Number of children under 5 years per one thousand women between 15 and 45 years of age in the United States from 1800 to 1930.

number of births per thousand of population per year, while the death rate will be low. Such a population would, on the face of the statistics, seem destined to overrun the earth. Fifteen years from now, however, the birth rate will have sunk almost to zero, for the original couples will be mostly past the reproductive period while their children will not yet be old enough to take up the burden. At the same time there would be an appreciable death rate. With births at zero, and deaths at any positive figure, such a population would seem to be headed for extinction. Neither of these apparent conclusions is correct; the birth and death rates are meaningless because of the unusual age distribution.

Though no population is of such extremely irregular distribution as to age, every population in which either birth rate or

death rate is undergoing a change will present some irregularities. After birth and death rates have remained constant for a long time the population becomes stabilized, and then the relative rates of birth and death are truly indicative of population growth. When births equal deaths and the population is stationary, the age distribution will be as shown in Fig. 155, left. How different the population of the United States was in 1930 is indicated at the right. The notable features of this latter distribution are (1) the much broader base of the cone for the United States as compared with a stabilized population (due to high birth rate and immigration in the past), and (2) the smaller number of children under 5 years as compared with those between 5 and 10 years (due to the recent decline of the birth rate). How

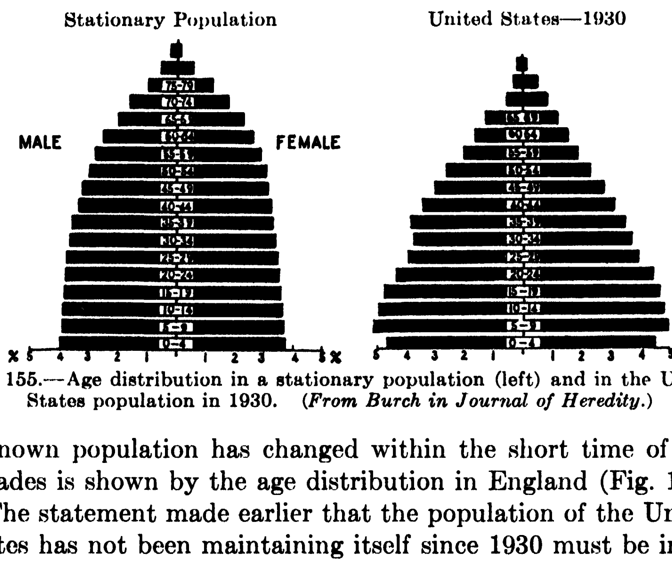


Fig. 155.—Age distribution in a stationary population (left) and in the United States population in 1930. (*From Burch in Journal of Heredity.*)

a known population has changed within the short time of two decades is shown by the age distribution in England (Fig. 156).

The statement made earlier that the population of the United States has not been maintaining itself since 1930 must be interpreted in the light of the age distribution. While the population is *now* growing, a stabilized population exhibiting the same properties would actually be declining. Many vital statistics have led to wrong conclusions because of the failure to adjust them in relation to a population of normal age distribution. The crude birth and death rates must always be translated into "true" or stabilized rates before being used for long-range prediction.

Probable Course of Population in the United States.—The peak of births in the United States, as already indicated, occurred in

1921. What has happened since is a remarkable decline. What will happen to it in the future is largely conjectural, though there are many grounds on which to base estimates. The death rate has dropped considerably since 1900, and the opinion is prevalent that it cannot fall much farther in the near future. The remaining factor which enters into future population, namely, immigration, is under control by law, which can be extended if desired; at present it is very low. Taking all these elements of growth into consideration, one could predict what the course of population will be. This has been done by various writers, with discordant results. Even the same authors may reach several different conclusions.

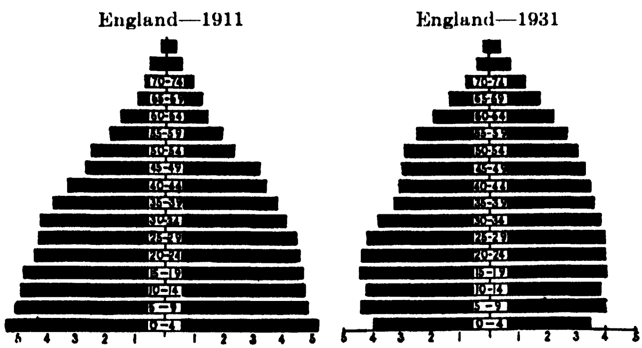


FIG. 156.—Change of age distribution in the population of England from 1911 (left) to 1931. (*From Burch in Journal of Heredity.*)

The older work of Pearl and of East on this question has already been noted. One of the more objective recent studies (objective in that several methods have been used) is that of Thompson and Whelpton (1933). Among the possibilities which they foresee are the following. A population of 202,000,000 may be reached by 1980; or the maximum may be only 138,000,000, reached by 1955 with a decline thereafter to 129,000,000 in 1980; or a population of about 155,000,000 may be attained by 1980. Modifications of these results arise out of different assumed birth, death, and immigration rates and altered expectation of life. The authors appear to regard the last result as the more probable. O. E. Baker chooses however to adopt the more pessimistic view that, after 10 or 20 years of slow increase and a few years of equilibrium, a decline will set in.

The presence among these possibilities of a decrease in population after a maximum has been reached introduces a further

unusual element. A declining population includes fewer dependent individuals than does either a stationary or growing one. The reduction occurs among the children and is partly offset by a relative increase of the old. The economic effect of such a change could be considerable in spots.

From Farm to City.—When agriculture was the chief occupation of civilized people, the populations of most countries were governed by similar principles. With the growth of cities the problems have become differentiated. In the United States in 1820 about 72 per cent of the population was agricultural, and by 1870 still about 54 per cent were on farms. With the industrialization of the country the decline of the farm population was

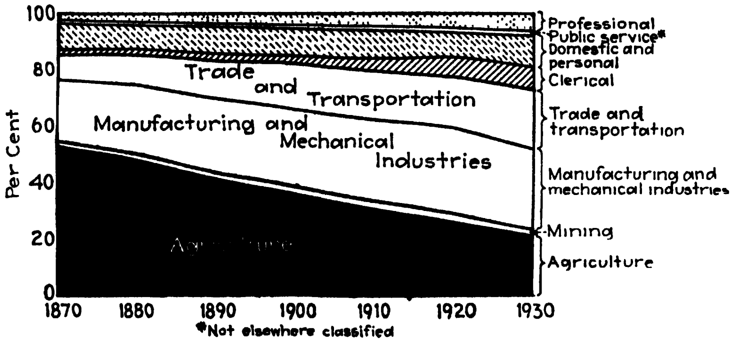
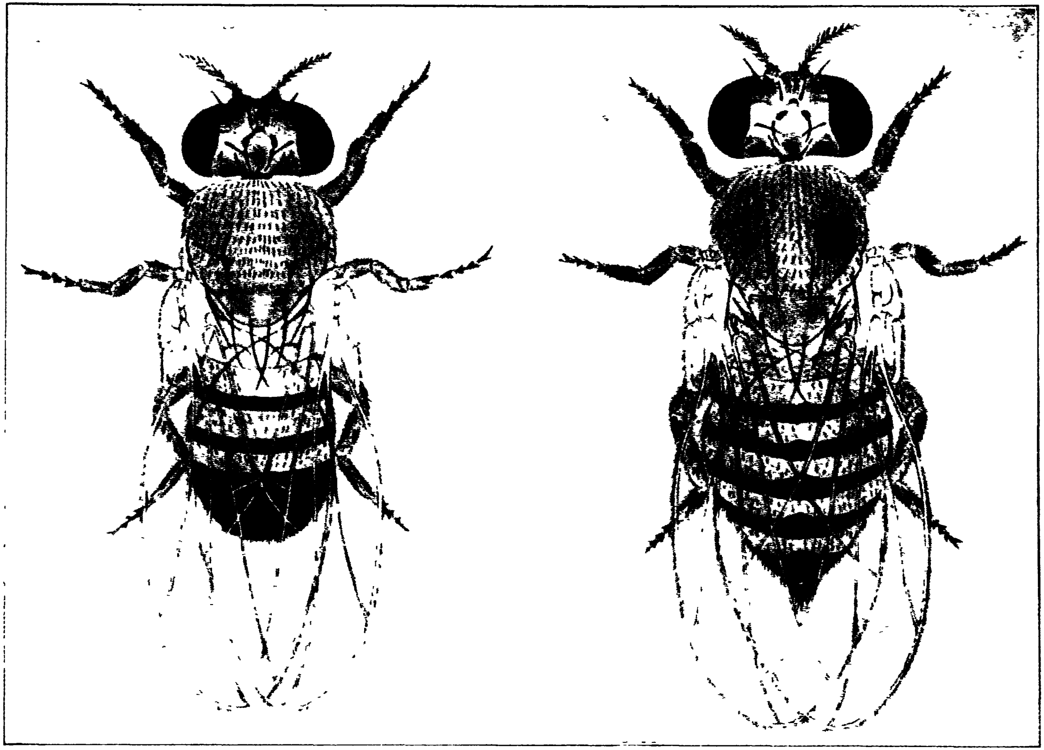


FIG. 157.—Decline of rural and increase of urban population in the United States from 1870 to 1930. (*From Holmes, Human Genetics and Its Social Import, McGraw-Hill Book Company, Inc.*)

accelerated, and in 1930 only 21 per cent of the nation was agricultural. During this latter decline of farming, manufacturing increased moderately, commerce more than doubled, and domestic service remained nearly stationary (Fig. 157). Farmers were moving to the cities to take the industrial and commercial jobs which were being created. At the same time there was a movement back to the farms, in which mostly elderly people participated.

The farm-to-city movement took young people to urban environments at the age when families are being reared, or earlier, and partially replaced them with others past the reproductive period. The effect of this exchange *should* be to raise the birth rate of cities and lower that of the farms, and some cities do actually show a higher crude birth rate than the rural



FRONTISPIECE.—The vinegar fly, *Drosophila melanogaster*, male (left) and female. This species of fly has developed hundreds of new modifications affecting eyes, wings, legs, body color, spines and physiological characters. For this reason, and because the generations pass rapidly and the flies can be easily reared in large numbers on food that can be furnished the year round, it has been extensively bred in laboratories by students of heredity. More has been learned concerning heredity from this one species since 1910 than had been learned from all sources before that time.

areas have. High city birth rates are occasionally created by the unfortunate practice of crediting all births in a maternity hospital to the city in which the hospital is located, rather than to the communities from which their patients have been drawn. But even when the somewhat higher birth rate in a city is all due to the residents of that city, this excess is deceptive because of abnormal age distribution. When the birth rate of a city is based on the number of women of child-bearing age, not on the total population, the corrected rate usually falls below that of rural regions. Actually cities on the average have a distinctly lower true birth rate than have the farms.

For some reason cities have generally managed to reduce the birth rate of all classes who come to dwell in them. The reason may be economic, since children on the farm help with the work, while in the city they are a burden on both the time and the financial resources of the parents. There is probably also some element of sophistication in the rural-urban difference. But whatever the cause, cities have been, as Holmes (1936) puts it, "centers of extinction." Farms furnish the real elements of population growth. The extent to which cities fail to maintain themselves is indicated by the true or stabilized birth rates: cities of New York state 27 per cent below replacement requirements, those of Illinois 23 per cent below, those of Massachusetts 18 per cent below, those of Vermont 11 per cent below, those of Indiana 7 per cent below, and those of Maine 5 per cent below (Lorimer and Osborn 1934).

From the standpoint of mere population maintenance, therefore, the accumulation of large numbers of people in cities is an unfavorable development. How the *quality* of the population is affected by this drift to the cities belongs rather to the discussion of eugenics but should be mentioned here. Beginning with Francis Galton, it has generally been held that the mentally better equipped leave the farms to seek better fortunes in the urban centers. It has never been possible to prove this assumption adequately and directly, and quite possibly the recent trends of farm management, tending to make rural life more attractive, have reduced the distinction. Cities may not have a much higher mental level (though intelligence tests have generally indicated that they do) for in them collect also the tramps and loafers. But if it is true that the superior parts of the farm

population go to the city, and the city reduces their fertility, urbanization appears doubly undesirable.

Course of Population in Other Countries.—A number of other countries are experiencing the same decline in the net rate of population growth that the United States has begun and faces for the future. They have largely preceded America in this decline and are indeed one of the reasons for predicting a similar change for this country. Though all of these countries still have more births than deaths, correction of their indices for age distribution leaves some of them a substantial deficit. England and Wales, for example, have an excess of births over deaths amounting to 2 per thousand of the population; but the adjusted rate of these countries is −3, or 3 per thousand less than maintenance requirements. Sweden has almost as great a deficit, for Germany and Switzerland the index is below −2, for Norway below −1, and for France about −0.4. It is of interest that, as between German and French population growth, a contrast long fraught with political significance, the French have at the moment a long-range advantage.

Other countries of Europe are still increasing. For Holland and Italy the stabilized rate of increase is over 4 per thousand, for Poland and the Ukraine over 7 per thousand. The Slavic peoples of Europe are increasing about twice as rapidly as the Germanic. Italy, despite her recent strenuous efforts to maintain the fertility of her women, is gaining only about as rapidly as Holland with reputedly one of the best-organized systems of birth control in existence. How far knowledge of these situations will modify the attitude and practice of the peoples involved only time will tell. So far governmental attempts at regulation and propaganda have not been eminently successful.

The crowded regions of the earth are India and China, and respecting them there is no reliable information relating to births and deaths. Japan has long been regarded as overpopulated and still growing rapidly, and these conditions have been advanced by Japan herself to justify her military incursions into China. According to Penrose (1934), however, the Japanese people are not extraordinarily fertile. Their highest recorded birth rate—at what date is not indicated—is less than 35 per thousand, and the present population seems to have been attained by birth and death rates similar to those which obtained in Europe. Penrose

believes that contraception is being practiced, that for the past dozen years or more the birth rate has fallen rapidly, and that the annual births will number about 2,100,000 for the next two decades. On the basis of these figures he adopts the conclusion of Uyeda that Japan's ultimate population will be about 80,000,000, reached by 1950. The oft predicted 100,000,000 he holds cannot materialize. How much the further industrialization of the country may affect these figures is unknown, but some further drift to the cities is expected. Most western writers have

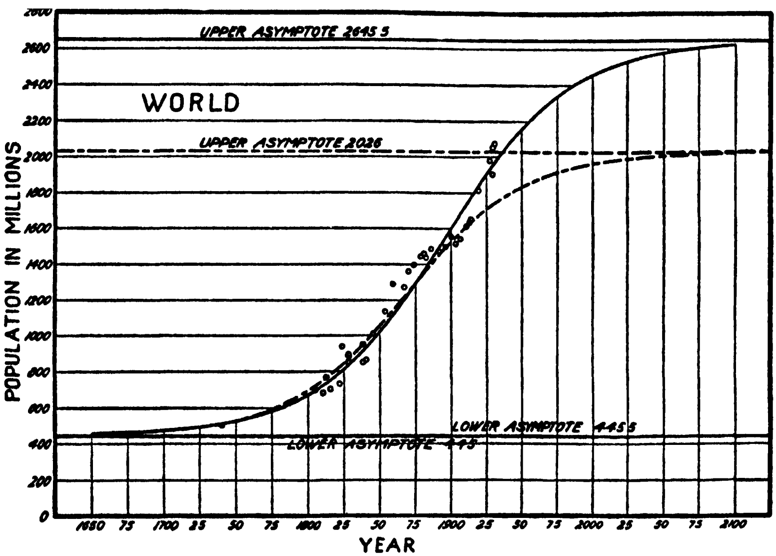


FIG. 158.—World population, with predictions for the future, based on data of 1914 (broken curve) and of 1931 (solid curve). (*From Pearl and Gould in Human Biology.*)

expressed the view that Japan's industrial progress will be somewhat restricted by her lack of inventiveness, and population growth could be affected adversely by this characteristic.

World Population.—So many countries take no census and report no births or deaths that an estimate of world population is difficult. Pearl (1936 *a*) has nevertheless essayed the task of making one, and considers 2,074,000,000 a probable figure. This is a rise of 200,000,000 above what was regarded as a good conjecture a decade or so ago. Pearl also places the annual increase at 27,000,000, an 80 per cent rise from the same author's

earlier estimates. These are assumed absolute numerical values, and therefore comparable to the net rates of increase, or births less deaths, already given in earlier statistics. They are accordingly subject to the same correction for age distribution which has been applied to other more limited populations. However, since the data on which a correction would have to be based are not available, Pearl and Gould (1936) use the logistic curve (page 321) to estimate the ultimate population of the earth. The projected curve indicates that by the year 2100 the number of people will be near the upper limit (Fig. 158) of 2,645,500,000.

Danger Spots.—The existence of overcrowded areas like those of India, China, and Japan and at the same time underpopulated regions such as Australia, New Zealand, and portions of North America is doubtless a temptation to aggression. The white race, long before any crowding existed, had visited and claimed large parts of the earth. The incentive at first was partly adventure, partly a belief in natural sources of wealth in other parts of the world, but not lack of room to lead an agricultural existence. Then came an enormous expansion in numbers, and the urge to control more of the wealth of the earth became overpowering. An industrial nation can support a large population only if raw materials are available, and as a rule no one limited area provides all of them. Trade would secure all these things, but as jealousies arise trade—so the argument goes—has to be protected. Military strength enables a nation to acquire new lands by force, and added territory requires—so again goes the argument—more military strength. For military strength more population is needed. Even where there are already too many people, governments are calling for still more, in order to protect seized lands, so that population may increase, and make more protection, greater military strength, and hence more population necessary.

There is little doubt that among the aggressions that have startled and depressed civilization in recent times, some have resulted primarily from overcrowding. But is that the only or chief cause? The worst overcrowded nations (India, China, for example) are not the ones guilty of forcible seizure, but their lack of aggressiveness may perhaps be attributed to lack of opportunity. The captured territory sometimes will not afford any relief of the kind sought, but here ignorance may be responsible.

There is on the whole enough lack of correspondence between aggression and actual relief gained from it to raise the question what else may serve to initiate international pillage. It is possible that ambitious and demagogic rulers ride to power and continue themselves in it on the passions aroused by their propaganda, knowing full well that the prospects held out by them will not be realized.

Reasons for Limitation of Population.—Reasons which make restriction of the population desirable must be distinguished from the causes which have brought it about. Among the former the limited food supply has usually been regarded as foremost. Agriculture cannot supply food for all the human beings who might conceivably find room on the earth. East has examined the world food situation and concludes that the prospects of great additions to it are not bright. In one of the crowded countries, Japan, Penrose holds that local production will be great enough in *quantity* to supply the ultimate 80,000,000 who will occupy the land but that in quality their diet is likely to be deficient—lacking in proteins and vitamins. For an industrial nation, exhaustion of irreplaceable natural resources is also a possibility which must be faced. Essential minerals, for example, if not in unlimited supply, may limit population unless substitutes can be found or unless trade can be indefinitely maintained.

The manner of bringing about such restriction as will fit the population to its means of sustenance has varied. Disease and hardship used to balance a high birth rate with a high death rate, but with the advance of medical practice this source of restriction has been greatly reduced. The later limitation through a declining birth rate is not so well understood. Since through contraceptive practices the sex impulses can now be largely dissociated from their natural consequences in reproduction, the reduction in the size of families must be in some other way psychological. Popenoe (1936) in a study of childless marriages discovered a number of motives. Whether such motives are correctly stated on inquiry could be questioned, for many people do not know the reasons for their own behavior. There is a possibility of unconscious rationalizing. However, his clients could hardly be accused of painting a too rosy background for their avoidance of parentage, for the one they did portray was a drab one. Most of the motives advanced were selfish; only 1

person in 20 gave a eugenic reason. Holmes (1936) makes two generalizations about the decline in the birth rate: (1) as people become wealthy they tend to restrict their families; (2) people limit their families for economic reasons. The wealthy find it inconvenient and a drag on their social activities to have many children; and the less well-to-do are unable to rear large families in keeping with accepted standards. The attitude of certain religious organizations has in the past tended to restrain their members from practicing birth control, but there are signs that this influence is being discarded and that it is becoming ineffective anyway.

In addition to these economic and personal reasons for family restriction, some writers believe there is infertility of a genetic nature which is on the increase. Some think there is a cycle of fertility: primitive races are fertile, but, as civilization advances, there are degenerative changes including reduced fertility which lead to extinction. It is doubtful whether either of these views is correct apart from psychological and physiological phenomena which have a more direct explanation. The general health and vigor of a people should influence fertility, and, if there is any decline of vigor, there would probably be a decline of the birth rate. The sex impulses bear some relation to the degree of other activities, becoming more active in periods of leisure and relaxation, and declining with mental strain and worry. In the absence of effective birth control, as in the past, the difference between rural and urban birth rates could be partly explained in this way. But it must be admitted that the whole story of the differential and declining birth rate has not yet been told.

Optimum Population Density.—If peoples should ever decide to try to control their numbers, reduce them as they exceeded proper bounds, bolster them as they dropped to dangerous lows, it would be desirable to have some formula by which the most advantageous size of population could be ascertained. No such formula has been developed; whether one can be devised is questionable. A general principle might well be that population should be maintained at that level which would bring the greatest good to all. The number should be above a certain minimum, to make full use of the advantages of division of labor. It should likewise be below a maximum at which natural resources prove inadequate. Health in relation to modes of life, and

opportunities for medical care and schooling, also enter into the calculation, partly on opposite sides of the equation. No one knows what the minimum and maximum are, but Burch (1937) suspects we are well beyond the maximum in many parts of the United States at the present time.

Any corrective measures designed to control population should certainly be selective and, as such, belong to the eugenics movement. So far as they concern the birth rate, they have been discussed in the preceding chapter.

CHAPTER XXIX

RACE PROBLEMS

A race is a group of people having many characters largely in common because of common descent. The real basis of judgment of race is the possession of like genes. Not all of the genes are common to all members of a race, for there are individual differences within races just as within species. It is not necessary, indeed, that all individuals have any one gene in common. The characters which distinguish races are all multiple-gene characters, so that one gene in one individual could be different from the homologous gene of every other individual, without so altering him that he would not be recognized as belonging to the same race. Each locus could have a gene which in one individual differed from its homologues in all the rest of the population, without destroying the general similarity which characterizes races. All that need be true is that most members of a race have most of their genes in common. This flexible requirement allows a wider range of variation in some races than in others, and such differences actually exist. A race is not a fixed group, and the word race does not everywhere apply to comparable assemblages. Such flexibility of the term may not be desirable, but it cannot be helped.

The only likely way in which thousands or millions of individuals may come to possess genes in common is by inheriting them from the same ancestry. It is *conceivable* that genes derived from different ancestries might come to be alike through convergent mutation. It is also *conceivable* that enough genes from different sources might mutate in this convergent fashion to build up two unrelated groups of great similarity. Whether two such groups would be regarded as belonging to the same race, if their separate origins were known, is a question which geneticists have not had to answer, though taxonomists have sometimes thought they were faced with that situation in species. Because the amount of convergent mutation necessary to make

two unrelated groups look like the same race is so exceedingly improbable, the stipulation in the definition of race that the likenesses must be due to common descent is justifiable.

Nonracial Characters.—Since certain characteristics are developed in people by the environment, it is necessary to take some precaution to exclude them when race is under consideration. Of the physical characters, size and proportions of the parts of the body are partly environmental, being responsive to diet (vitamins) and to the hormones which are themselves responsive to diet and other conditions of life. Some confusion regarding races arises out of these external influences.

A much more common error concerning race is to confuse it with language, or nation, or even religion. People living together learn to speak the same language, regardless of their genes or descent. They also live under the same scheme of government merely because they are neighbors. Neighbors tend to adopt the same customs and to respect the same traditions, without reference to remote ancestry. And so, there is no Anglo-Saxon race. Those who use that expression may have the correct concept of race in mind but are mistaken in applying the term to a particular group. The people they call Anglo-Saxon have a certain speech, a body of traditions, and a group of customs in common; but racially they are very mixed. There is no French race, for the inhabitants of France have derived their genes from three somewhat distinct sources.

Racial Classification.—Mankind falls into three great groups, with several minor ones which elude simple classification. The three large groups are the Negroid, the Mongoloid, and the Caucasian races. Each has its subdivisions. In the Negroid group are the African Negroes, the Oceanic Negroes, and the Negritos or Dwarf Blacks. Closely related to the last of these are the Bushmen of South Africa. In the Mongoloid group are the Asiatic Mongoloids, the Oceanic Mongoloids, and the American Indians. Allied to the Oceanic Mongoloids are also Polynesians. The Caucasian race is composed of the Nordic, Alpine, and Mediterranean races, with the Hindus similar to the Mediterranean.

Among the minor races not easily placed in any of the major assemblages are (1) the Ainu of Japan, with leanings to the Caucasian type, (2) the Australian aborigines who are nearer

the African Negroes but different from them, and (3) the Indo-Australians who approach the Hindu.

The classification of races goes farther than this and is extended later in this chapter; but the main features here indicated will furnish the orientation needed for a first discussion of race problems.

Characters Distinguishing Races.—Anthropologists have always placed emphasis on stature as a racial mark, though it is subject to much fluctuation, partly environmental, within each race. Since few races have an average stature of less than about 60 inches, and none a mean of over 70 inches, it is clear

FIG. 159.—Skull shape as a racial character: left to right, narrow, medium, and round. (*From Ward's Natural Science Establishment.*)

that there is a great deal of overlapping. Stature alone would never serve to determine the race of an individual, though it might almost exclude certain races.

The cephalic index, or ratio of width to length of the skull (Fig. 159), is also very extensively used. It has the fault of responding somewhat to dietary or other conditions. Descendants of European immigrants in America have for this reason been found to differ from their relatives in Europe. In some races the head became narrower in America, in other races broader. In a homogeneous environment, however, head shape is a very serviceable racial mark.

The nasal index, or width of the nose in relation to its length, is likewise racial. In the Negroid races that ratio is very high.

Prognathism, or the protrusion of the jaw in front, is another trait the anthropologists employ.

Cranial capacity varies considerably. For the white race as a whole, the average capacity is between 1450 and 1500 cc. in males, about 10 per cent less in females. European males have a mean between 1500 and 1600 cc., American Indians between 1400 and 1500 cc., and Bushmen between 1300 and 1400 cc.

Texture or shape of the hair is important. As indicated earlier (page 251), this shape is due to the form of the hair in cross section. The woolly hair of the Negro is considerably flattened, the straight hair of Mongoloids circular, and the wavy hair of Caucasians intermediate.

Hairiness is especially useful in some situations. The variation between races chiefly relates to the beard and body hair. Caucasians are usually quite hairy, while Mongoloids and Negroids are relatively smooth. The Ainu are separated racially from the Japanese largely because of their hairiness. Likewise the Australian aborigines are considered a different race from the Negroid chiefly because they are hairier.

Hair color and eye color are useful because they are almost free from nongenetic modification. Black hair and eyes are the rule outside the Caucasians but within this race the color has many gradations.

Miscellaneous Race Distinctions.—In addition to the easily observed characters which anthropologists use, a number of other differences between certain races have been observed. The blood groups are differently distributed. Most American Indians are of group O, possessing neither agglutinogen A nor B (page 108). The proportions of A and B in other nationalities vary from west to east in the Old World, A being prevalent in Europe, while B gains to the eastward (Fig. 160). At first it was suggested that the absence of any agglutinogen was the primitive condition, that A arose by mutation in Europe, B by mutation in Asia, and that each spread from its place of origin. Some later racial studies show, however, that the Poles and Russians are similar in their agglutinogens to the South Chinese and the Japanese and that the North Chinese and Hindus are nearly identical (Kroeber 1934). These similarities could hardly arise out of close kinship, hence probably B arose more than once as parallel mutations (page 242) in different races.

Basal metabolism was found to be 6.5 per cent higher in Mayan men in Yucatan than in Caucasians, in south Indian women 17.4 per cent lower than in white women, and in Australian aborigines 14 to 16 per cent below Caucasians (Benedict 1932). If these differences stood alone, they might be attributed to climate and food; but American-born pure-blooded Chinese girls in Boston had a metabolism 9 per cent below that of American white girls, and here climate was identical and food not very different.

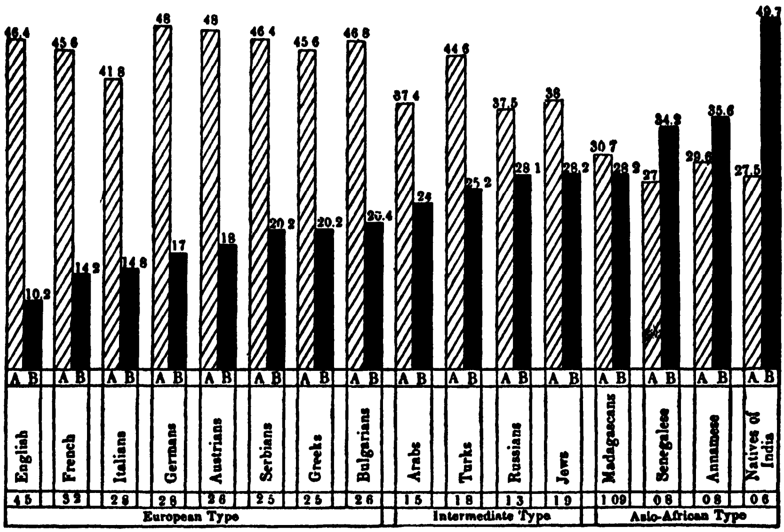


FIG. 160.—Diagram showing the increase of agglutinogen B of human blood from west to east in Europe and Asia, and the decrease of agglutinogen A in the same direction. (*From Snyder, Blood Grouping in Relation to Legal and Clinical Medicine, Williams and Wilkins Co.*)

Sweat glands are more abundant in Negroes than in whites, at any place on the body, though their relative distribution is the same in both (Glaser 1934). Musical discrimination is said to be keener in yellow and black races than in whites; tests that suffice for the last are not refined enough for the first two. Negroes have been more susceptible to tuberculosis than whites, though they appear to be overcoming it (Holmes 1934); they are still more handicapped by venereal disease. Negroes in the World War were found (Davenport and Love 1921) to exhibit such minor differences from the white troops as longer

arms and legs, shorter trunk, narrower pelvis, shorter neck, and broader shoulders.

These various distinctions would not aid an anthropologist greatly in diagnosing races, but they all help to illustrate that races are distinguished by hereditary characters, that is, by genes.

Three White Races.—Because the civilized peoples are prevalently Caucasian and America is mainly European, three

FIG. 161.—Alpine woman from Lapland. (*From Mjøen in Eugenics in Race and State, Williams and Wilkins Co.*)

FIG. 162.—Mediterranean type from south Italy. (*From Dixon, Racial History of Mankind, Charles Scribner's Sons.*)

of the subdivisions of the white race figure extensively in the topics further developed in this and the next chapter. The Alpines (Fig. 161) have a round face and wide skull, moderately dark hair and eyes, and a stocky build. They are found chiefly in the divisions of old Russia, in the Balkan states, Switzerland, Bavaria, Austria, northern Italy, and central France. They are thought to have come into Europe from Asia, past the Black Sea. The Mediterraneans have a slight build, narrow skull, and dark or swarthy complexion (Fig. 162). They are in

Portugal, Spain, southern Italy, Sicily, and north Africa, and less extensively the British Isles and littoral regions of the Balkan peninsula. They too appear to have come from Asia, but through northern Africa and across the Mediterranean Sea. The Nordics (Fig. 163) are tall and fair, with light hair and eyes, narrow skull, hairy body, and a domineering disposition. They are collected largely around the Baltic Sea in Scandinavia,

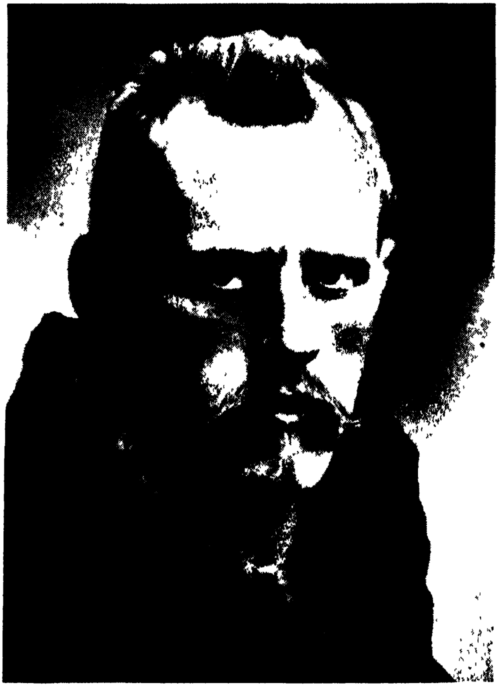


FIG. 163.—The Nordic type. (*From Mjøen in Eugenics in Race and State, Williams and Wilkins Co.*)

Germany, the Netherlands, and the British Isles, but have some representation at many other places. They have not been definitely traced to any source outside of Europe.

These definitions must not be interpreted to mean that pure races exhibiting the characters named exist in the regions indicated or anywhere else. They represent idealized races and doubtless more accurately the conditions of the ancestors of the present-day groups. Much mixture between them has occurred so that no white race is now pure. The nearest approaches to

purity and adherence to "original" type in the white race are found in Sweden (Nordic) and Sicily (Mediterranean).

Mentality of Races.—Because of its importance in problems of race and immigration in the United States, many attempts have been made to determine whether Negroes, American Indians, Mexicans, and the various European nationalities differ notably from American whites and from one another in native ability. The decision has usually to be made by use of the several intelligence tests, and it must be admitted that no test yet devised reveals merely natural capacities. They all measure experience in part and are to that extent unfair to the individual or group tested in a strange environment. Studies by Garth gave the full-blooded American Indians an IQ of only 68.6, Texas Negroes 75, and Mexicans 78. The samples tested were each over a thousand individuals. A test of Southern Ontario Indians gave them a score of 97 on the Pintner Nonlanguage Test, 92 on the Pintner-Paterson Performance Tests, only 80 on the National Intelligence Test, and still lower on the Pintner-Cunningham Primary Test. A number of investigations, and some army tests made at camps during the World War, showed that mulattoes made higher scores than pure Negroes, and that the ratings rose as the color lightened. There is, however, much geographic variation in the Negro scores. The score of Northern Negro recruits in the World War was about the same as that of South Central whites but far above that of South Central Negroes. Whether this is due to selective migration, or to difference in educational advantages, is not clear. Probably both are involved. Admixture of white blood is also related to professional and commercial success of the Negro. College graduates, lawyers, doctors and writers of Negro extraction are mostly mulattoes. The 145 life members of the National Business League included 71 light mulattoes, 60 medium mulattoes, and 14 Negroes. Most writers have taken these comparisons more or less at their face value and have attributed the greater success of the mulattoes to their white inheritance. One suggested that during slavery the superior women slaves became the mothers of half-breed children, and that since then the mulattoes have tended to intermarry and to maintain a separate group retaining the original superiorities. On this theory the higher ratings and success of mulattoes could be as much dependent on

the Negro parentage as on the white. The tendency of mulattoes to keep apart from pure blacks is confirmed on other grounds; the selectiveness of slave-master matings is highly questionable. And whatever race crosses have been effected since emancipation have probably involved the worse elements of both races.

The Mexicans in the United States were apparently selected for immigration on an unfavorable basis. Since no check is made of Mexicans entering this country, the states from which they are derived have been determined by Gamio from a count of the money orders sent to Mexico from New Mexico, Arizona, and California. The results indicate that the immigrants are of the peon class forced to emigrate by economic conditions, as could well have been guessed by the characters of the migrant group. Intelligence tests of Mexicans in the United States are therefore based on the poorer classes and are not representative of the nation. Low scores are accordingly to be expected.

European nationals have been studied in various ways as they applied for admission to the United States. Kolb (1932) used the Healy Construction Test "A" on literate adult males of five nationalities, and obtained the quickest performance from the Norwegians, followed by Germany, England and Wales, Irish Free State, and South Italy. More extensive comparisons were obtained in the examination of foreign-born recruits in the World War. The high ratings (A and B) and the low ratings (D, D –, E), were distributed as in the table on page 345.

School children of various national-racial groups in Massachusetts mill towns were given the Pintner-Cunningham Test (first grade), Dearborn A Test (second and third grades), and Dearborn C Test (sixth and seventh grades) and were ranked in the following order, highest first: Polish Jews, Swedes, English, Russian Jews, Germans, Americans, Lithuanians, Irish, British-Canadian, Russians, Poles, Greeks, Italians, French-Canadians, and Portuguese.

The European groups referred to are necessarily national ones, but they have some general racial differences which make them of interest. While the tests appear to show racial differences in mental qualities and while such distinctions are as much to be expected as are physical ones, opinion differs regarding the validity of the comparisons. Some anthropologists hold that such differences as exist are all environmental, but this

should not be true if mental traits are in any degree inherited. A reasonable attitude would seem to be that racial differences in ability are to be expected but that intelligence tests without bias have not yet been invented to discover them.

Results of Intelligence Tests of Foreign-born Recruits in United States Army

Country of birth	Percentage attaining	
	Grades A, B	Grades D, D −, E
England	19.7	8.7
Scotland	13.0	13.6
Netherlands	10.7	9.2
Canada	10.5	19.5
Germany	8.3	15.0
Denmark	5.4	13.4
Sweden	4.3	19.4
Norway	4.1	25.6
Ireland	4.1	39.4
Turkey	3.4	42.0
Austria	3.4	37.5
Russia	2.7	60.4
Greece	2.1	43.6
Italy	0.8	63.4
Belgium	0.8	24.0
Poland	0.5	69.9

Race Hybridization.—One practical problem around which questions of comparative racial values tend to revolve is that of the desirability or undesirability of race crossing. A popular conclusion has been that hybrids between races are apt to sink to the level of the inferior race or to be worse than either of them. With respect to characters determined by environment, that conclusion is often justified, particularly in regions where miscegenation is regarded with disfavor. In such regions hybrids are social outcasts, and their status may tend to unsettle them. Hybrids may also be inferior for genetic reasons, because where race mixture is taboo it is chiefly the inferior individuals of either race who will indulge in it. In the latter situation the offspring may not be inferior to the children which would have

been produced by the same parents had each one married a similar person within his own race.

Where miscegenation is not frowned upon, as is true in most of South America, the value of race hybrids may be estimated on purely biological grounds. In size, color and some other characters, the hybrids tend to be intermediate, as was found by Davenport and Steggerda to be true of the brown people of Jamaica. If one race is superior to the other, an intermediate hybrid is inferior to one parent, but an improvement upon the other. To the race as a whole, there is no detriment in this relation. When two unequal races cross illegitimately, with the men coming from one race, the women from the other, the biological effect depends on which race furnishes the women. The women control the number of children, and hybridization is at the expense of their race. If whites are superior to Negroes and mulattoes first arose from unions of white men with Negro women, their intermediate mulatto offspring, supplanting an equal number of pure Negro children, should be an actual gain.

The principal biological disadvantage that may come from race mixtures is the production of disharmonious combinations. A favorite example is the combination of teeth and jaws of different sizes from different races. Within each race growth of one part is properly adjusted to that of the other, and well-formed rows of sound teeth are usual in purebred races. In the hybrid, however, with independent inheritance of size of jaw and size of teeth, small teeth may be set in a large jaw with consequent spacing, or large teeth in a small jaw resulting in distorted rows of teeth. There are numerous examples of such misfits in the tremendously hybridized American population, and their explanation may be as simple as just stated. Davenport adheres to this view, but Castle believes that conspicuous disharmonies are uncommon if the crossed races are not too dissimilar. Mjøen cites a number of misfit characters among Norwegian Nordic-Lapp hybrids, but some of them, such as small ears, will probably not be serious handicaps to their possessors.

The normal result of free intercrossing is the disappearance of racial distinctions. With any tendency to avoid crossing even when the races intermingle, a considerable degree of separation may be maintained. With respect to the Negro and white races

in the United States, Holmes points out that miscegenation is less frequent than formerly, and that fertility of the mulattoes is relatively low, hence the two races are not likely to merge.

The antagonisms that sometimes exist between unlike races are probably not due to any of their biological characteristics, but to their social relations.

Racial Composition of United States.—In the year 1930, there were in the United States approximately 12,000,000 Negroes (including all mulattoes that did not "pass for white" in their communities); 332,000 American Indians (raised to 337,000 in 1938); 139,000 Japanese; 75,000 Chinese; and 45,000 Filipinos.

The remainder of the population belonged to the various divisions of the white race, greatly mixed. There is no accurate information as to the distribution of these whites among the idealized branches of the white race, but there is an estimate of the numbers derived from the several nations from whom immigrants have been received. This estimate was based on the 1920 census and other data, was made by a committee of the President's Cabinet, and was published in 1928. Owing to the manner in which the estimate was made, part of the population is attributed to ancestors who were already in America in 1790 (the colonial stock), the remainder to those who have since immigrated, together with their descendants (the postcolonial stock). The distribution according to country of origin, for all countries furnishing as much as 1 per cent of the total, is shown in the table on page 348.

Though Mexico is included in this table of whites, the population classed as "Mexican" by the Census Bureau is "not definitely white, Negro, Indian, Chinese, or Japanese." This Mexican group had increased to 1,400,000 by 1930. No other group experienced any wide swing in that decade, hence the percentages in the last column must be approximately correct at present. While the tables refer to nations, the data given are the best available clue to the representation of the white races in the United States.

Fertility of Component Races in United States.—In computing the probable future racial complexion of the United States, the fertility of the several racial types is of interest. The Negroes, as the largest group of nonwhites, claim first attention. According to Pearl (1936 *b*) the inherent fertility of white and Negro

women is about the same, within limits of probable error (see Appendix for significance of probable error). Contraception, however, is more effective as practiced by whites than among Negroes. There is accordingly a higher birth rate among the Negroes (18.79 per thousand as against 17.15 per thousand in the whites) in a group of cities. Greater wastage in Negroes, partly due to greater prevalence of venereal disease, compensates the higher birth rate, leaving the rate of increase about the same in the two races. The birth rate of the Negro decreased as he

APPORTIONMENT OF WHITE POPULATION OF THE UNITED STATES IN 1920, BY COUNTRY OF ORIGIN
(000 Omitted)

Country of origin	Colonial stock	Post-colonial stock	Percentage of total
Great Britain and Ireland	31,804	7,412	41 4
Germany	3,037	12,452	16 3
Irish Free State	1,822	8,832	11 2
Canada	646	3,391	4 3
Poland	9	3,884	4 1
Italy	.	3,462	3 6
Sweden	217	1,760	2 1
Netherlands	1,367	515	2 0
France	767	1,075	1 9
Czechoslovakia	55	1,660	1 8
Russia (in Europe and Asia)	4	1,657	1 8
Norway	75	1,343	1 5
Mexico	294	832	1 2
Switzerland	389	630	1 1

migrated north and from farm to city, changing a trend that had once been greatly in favor of the colored race.

The rate of increase for the other major races in America is probably best indicated by the number of young children in relation to the number of women who might bear children. The figures for the year 1930 are given in the table on page 349.

The data for the Negro agrees well with Pearl's conclusion above. The other colored races are reproducing about twice as fast as either white or Negro, according to these figures.

The immigrant whites and their descendants were once the despair of America because of their high birth rates. It was cal-

Children under 5 Years per 1000 Women of Child-bearing Age in United States, by Races, in 1930

Race	Children per 1000 Women
White	481
Negro	497
Japanese	824
Mexican	906
Indian	924
Chinese	1051
Filipino	1090

culated that the camel, having been admitted to the tent, was proceeding to kick its master out. A considerable change, however, has come over this situation in the last decade or two. The children of the immigrants, if not the foreign-born themselves, are adopting the American family pattern, congregation in cities is having the same effect on them that it has on American farmers, and the birth rate has rapidly declined. The median size of family for native whites of foreign parentage in cities was in 1930 only 3.19, that of native whites of native parentage 3.14. The number of children under 5 years per thousand women of child-bearing age in 1930 was 479 for the native whites, 489 for the foreign-born. The unconscious struggle for supremacy seems to be almost at an end.

Concerning particular foreign stocks the information is sketchy. The French Canadians are probably still the most prolific, the Irish in Canada only about as productive as the English. In Woonsocket, Rhode Island, however, the Irish increase somewhat more rapidly than the English. The French in Canada tend to form a closed social group, while the Irish in both Canada and the United States show a strong tendency to marry outside their own ethnic circle. What effect this has on the reproductive rate is conjectural. Italians are more fertile than Jews. Intermarriages between ethnic groups result in fewer children than do intraracial unions. Birth rate in all of them seems to be more dependent on economic conditions than upon ethnic origin. And so, while there are probably even now certain stocks which are gaining on the native population, the prospect that they will long continue to do so is greatly diminished.

Racial Effects of War.—Much concern has been felt relative to the effects of war upon the fate of races, and some of the humani-

tarian opposition to war unconsciously rests upon racial considerations. The argument commonly advanced against wars, on racial grounds, is that the fighting forces are made up of the superior members of society and that whatever deaths occur result in a lowering of the average of qualities among the survivors. Opposed to this view is that of certain philosophers who hold that war is a blessing in disguise because it is a means of enabling superior races to triumph over inferior ones, thus making for world progress. It is difficult to avoid the suspicion that both of these views rest upon concepts of war in the past, sometimes in the distant past, rather than of modern war. Ancient wars were waged upon peoples and extinction was not an uncommon aim. Under such circumstances, war could easily be the means of replacing inferior races with superior ones. In recent centuries, however, war has seldom led to anything approaching extinction of the vanquished people. Casualties in modern wars have varied from 5 to 15 per cent of the forces involved. Any effect upon racial values must, therefore, depend upon possible racial differences between those killed and the survivors. This means in part that it is dependent upon differences between the army and the civilian population.

Any difference between the military and civilian groups must depend on the manner in which the army is raised. Physically, it may be assumed, the army is, on the average, superior to civilians, because physically unsound recruits are not accepted, no matter what general method of creating the army is employed. Other qualities of the military force must depend upon whether conscription or voluntary enlistment is the method of obtaining recruits. Volunteers presumably possess qualities of daring or patriotism, although there are doubtless many unrecognized motives which lead to enlistment, and the character of the fighting in prospect probably determines which motives prevail. Conscript armies, on the contrary, should be strictly representative of the population as a whole except in physical characteristics. Under the conscript system, therefore, death losses in battle should result in a reduction of the average quality of the people in physical respects, but not in spiritual ones. Under the volunteer system, a lowering of both physical and spiritual values is to be expected. How great these reductions are, it is, of course, impossible to state in any significant way.

Opposed to these reductions in the average racial value are the death losses in the civilian population. Every war of any consequence entails hardships among the noncombatants, and the death rate is increased. The weaker members of the population are naturally the first to succumb, and their loss results in a corresponding rise in the average quality of the survivors. It is not to be supposed that this rise equals the reduction due to losses in battle, but it is an element often wholly ignored when the effects of war are discussed.

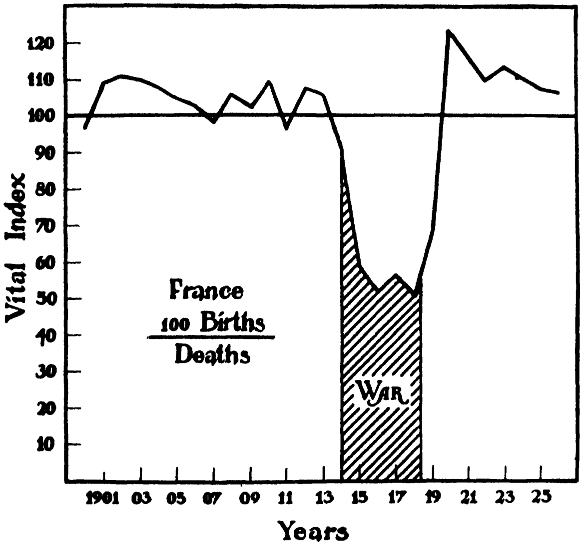


FIG. 164.—Curve of the ratio of births to deaths in France during the twentieth century, showing great reduction of this ratio in wartime. (*From Pearl in Human Biology and Racial Welfare, Paul B. Hoeber, Inc.*)

Another effect of war is the lowering of the birth rate due to the absence of married soldiers from home (Fig. 164). To whatever extent the army includes the superior, this reduction of the birth rate is presumably a loss. Publicists have sometimes advocated frequent furloughs with a view to maintaining the birth rate during war, but the result might be far from satisfactory. The mothers would no doubt be subject to the same hardships as the rest of the civilian population, and environmental injury to the children born could easily offset much of their inherent superiority.

CHAPTER XXX

IMMIGRATION

Immigration has affected the United States in various ways, economically, politically, and racially. Emphasis has shifted to one or another of these aspects as the number of immigrants increased or diminished and as the source of immigration changed from one section of Europe to another or to our nearer neighbors. Interest in immigration in connection with heredity centers mostly in questions of race.

Course of Immigration.—Immigration prior to 1820 was negligible. In that year a little over 8,000 immigrants came. Thereafter the number fluctuated, depending partly on political conditions in Europe but more on economic conditions in the United States. The first peak was reached in the early fifties, then the number fell until after the American Civil War. Immigration rose again in the early seventies but dropped with the depression following the panic of 1873. Industrialization was by that time in full swing and immigration rose sharply in the eighties but once more declined during the depression years around 1893. The all-time high was in 1907, when 1,250,000 immigrants entered this country. The average was maintained at 1,000,000 a year until 1914, when it dropped greatly during the World War. After the war immigration started up strongly, but by that time it was the subject of legislative action, and restrictions began to be imposed. Because of these restrictions the number of immigrants declined, and in the depression of 1932 and near-by years it was practically zero. Indeed, in certain years more foreigners left the country than entered it.

The first three peaks of immigration, ending with that between 1880 and 1885, were very largely due to arrivals from countries of northern and western Europe. The great influx between 1900 and 1914, however, was mainly from southern and eastern Europe. This change of the source of immigrants from northwest to southeast Europe played a large part in the adoption of

legislation governing immigration after the World War, the particulars of which will be pointed out presently.

Restriction of Immigration.—The attitude of the United States toward this century-old accretion of foreigners was at first one of welcome. The country was large and thinly populated and was devoted chiefly to agriculture. Additions of people of the same European stocks, who came here to settle on farms, could only be beneficial. It was not until the period of industrialization set in, around 1870, that this attitude changed. Immigration became an economic question then. The first restrictive legislation on national lines referred not to Europe but to the Chinese, the exclusion act being adopted in 1882. Three years later a law relating to contract labor was passed in the interest of American workmen. Other measures followed, all derived from economic considerations, which predominated until the early part of the present century. Then gradually the emphasis shifted, until now the biological reasons for restriction appear to be given greatest weight. As early as 1875 a law excluding prostitutes and certain criminals was adopted, which might be regarded as having a eugenic effect, but the exclusion was designed chiefly, if not solely, to remove a class that might become a social burden. Persons likely to become public charges were prohibited by law in 1882. The first statute having an obvious eugenic basis was that of 1917, which excluded more drastically than ever the defective classes and devised rules to enable officials to recognize potential defectives. In 1921, immigration from any European nation was limited to an annual increment equal to 3 per cent of the number from that country already here, based upon the census of 1910. Still greater restriction was provided in 1924, when the annual quota from each European country was fixed at 2 per cent of the number from that country who were here according to the census of 1890. The same law of 1924 made provision for a later change in the basis of the quotas, and after two legislative postponements this provision finally went into effect July 1, 1929. According to this provision, there may be admitted each year, from all European countries combined, a total of 150,000, which is to be divided among the several countries in proportion to the stocks derived from them already existing in the United States in 1920. Since the census of that year did not directly determine the

numbers of people derived from the several nations, these numbers were estimated by a commission from the President's Cabinet and the quotas established by presidential proclamation (page 347). Many more immigrants than the indicated 150,000 came in, since the law admits close relatives of citizens of the United States and since no country's quota is less than 100. There is no restriction against any American nation, and the immigration from Mexico has at times been heavy. The average annual total for a few years after 1925 was somewhat above 250,000.

Motive Leading to Present Law.—The shift of the basis of restriction from the 1910 census to that of 1890 and finally to the proportion of national origins was due to a belief in the Congress that countries of southeastern Europe were a less satisfactory source of immigrants than were the northwestern countries. A glance at results of intelligence tests of recruits in the army (page 345) shows the upper ranks to be held by England, Scotland, Holland, Germany, and the Scandinavian countries, while the Balkan states, Italy, Poland, and Russia placed low. If quotas were to be based on the proportions of the several nationalities already here at any given time, choosing the 1890 census would greatly restrict immigration from southeastern Europe, because up to that year not many immigrants had come from the southeastern states. A selective sifting of applicants on a national basis was thus effected without offensively naming any country outright as an undesirable source. Finally, if immigration was to be really proportional to the population already in America, numbers of immigrants would not suffice, but their descendants should be included in the determination. The calculation made by the President's Commission, already mentioned, relieved the restrictions on southeastern Europe somewhat, but still the numbers admitted from those countries were far below the admissions on the 1910 basis.

Laughlin's Studies of Custodial Institutions.—While the 1924 legislation was before the Congress, a study of the inmates of custodial institutions was made by H. H. Laughlin, with reference to the source of those inmates.

At the request of the Committee on Immigration and Naturalization of the Federal House of Representatives, he obtained information regarding the inmates of 684 state and federal

custodial institutions, with special reference to whether these inmates were of native or foreign stock. The quality of a given nation as a source of immigrants was judged by the relative frequency with which individuals from that nation were found in these institutions, taking into account the number of people from that nation in the whole country. For example, at the time when Laughlin's studies were made, there were 1,343,125 people of Italian birth in the United States. This number constituted 1.46 per cent of the total population of the country. If Italians are neither more likely nor less likely to furnish defectives than is the average of the whole population, 1.46 per cent of the inmates of the custodial institutions should be Italian. Occurrence of exactly the expected 1.46 per cent of Italians in institutions would be regarded as 100 per cent fulfillment of the Italian quota. Exceeding this 100 per cent of the quota is a bad sign; a figure less than 100 per cent indicates a more satisfactory condition.

For the immigrant population as a whole, without distinguishing nationalities or regions, Fig. 165 gives the most important information. Only white people are included, and they are divided into four classes: (1) native-born with both parents native, (2) native-born with one native and one foreign-born parent, (3) native-born with both parents foreign-born, and (4) foreign-born. Nine defects are listed. The horizontal black bands indicate the extent to which each class fulfills its quota in the custodial institutions, 100 per cent fulfillment being indicated by the vertical dotted line. Horizontal black bands extending to the right of this dotted line show an unsatisfactory record, while black bands not reaching the dotted line indicate a record better than the average.

The last group of four black bands shows that immigrants as a group, with their children, whether or not mixed with stock longer in America, get into the custodial institutions more than their share of times. The various defects do not share equally in this total, for in feeble-mindedness, blindness, deafness, deformity, and, to a less extent, epilepsy and crime, the foreign-born have a better record than the population as a whole. The low record of the foreign-born in the first four of these last-named defects is due simply to the fact that the defect is obvious, and such people are excluded; and in the last two defects, immigra-

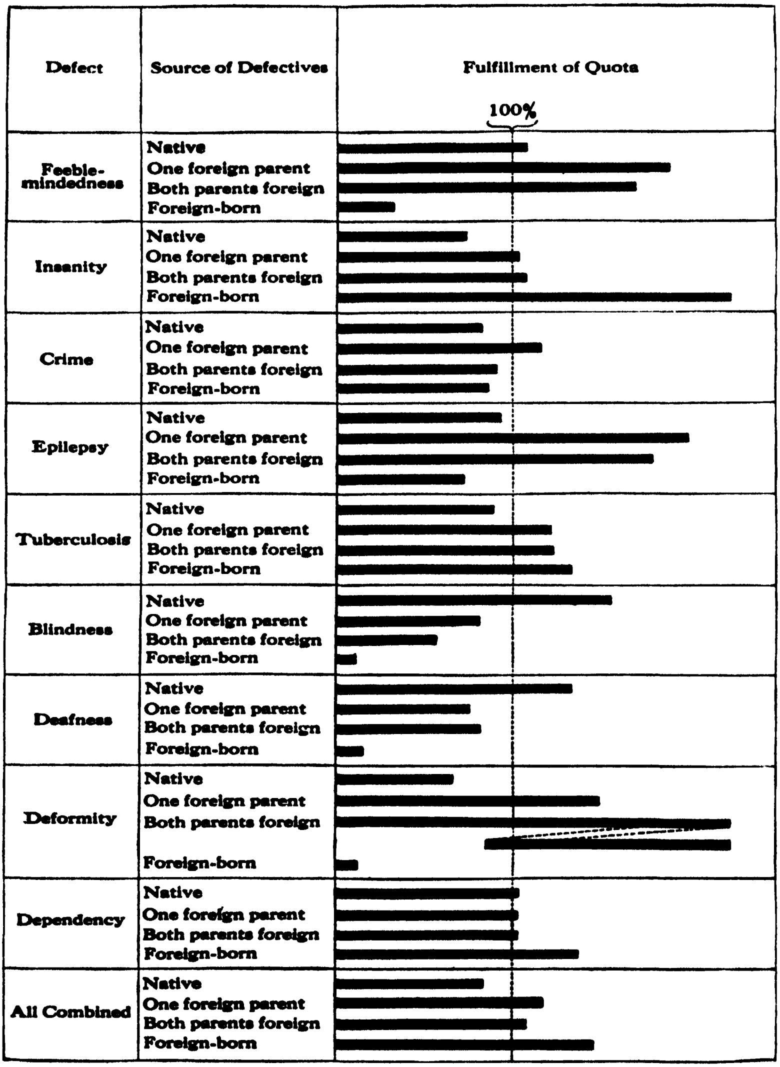


FIG. 165.—Chart showing the extent to which immigrants and their children get into custodial institutions in the United States, as compared with those who are native-born with native parents. The black bands indicate this extent. A black band extending to the right of the dotted line indicates that the group in question furnishes a larger percentage of the inmates of institutions than of the population of the country. Long black bands are therefore unsatisfactory, short ones very desirable.

tion officials have apparently been somewhat successful in detecting the condition. The good record of the foreign-born in these respects is more than offset by bad records in other characters, notably insanity. Since insanity accounts for over 43 per cent of the total number of inmates studied, not much help is needed from the tuberculous and dependent to make the total for the foreign-born shown in the last line far above their quota.

Two things in Fig. 165 are worth noting in particular. One is that the children of the foreign-born are, with respect to feeblemindedness and epilepsy, not nearly so satisfactory as their immigrant parents. The reason for this is presumably that the defects are both approximately recessive, and heterozygous immigrants were admitted because they were themselves normal. If their family histories could have been examined before admission, it is likely that most of these heterozygotes would have been excluded. The other particularly important feature of the figure is the very high percentage of insane among the foreign-born. Since their children have a much better record in this respect, there is room for the suspicion that much of the insanity of the foreign-born is not due to heredity. There is abundant reason for the assumption that insanity is due partly to environment. Jennings has pointed out that hardship during the period of adjustment to a new environment may easily account for part of this insanity. If environment, rather than heredity, is responsible for the excess of the quota in this, the most common defect, the last lines of Fig. 165 become somewhat less damaging to foreign nations as a source of our population. It is of course possible that the lower incidence of insanity among the children is due to the fact that insanity develops chiefly in middle life, and most of the children have not yet attained such years. This appears to be a not unlikely cause of at least part of the difference.

Northwest versus South and East Europe.—The preceding section, in so far as it really relates to qualities that are hereditary and hence racial, merely indicates the undesirability of any immigration that is unselected. The justification of the immigration law of 1924 and the national origins modification of 1929, which distinguish between northwest and southeast Europe in favor of the former, must be sought in a further analysis of Laughlin's data. Combining the countries of southern and eastern Europe, on the one hand, and those of northwestern Europe

on the other, one finds the defectives of our custodial institutions originating as shown in Fig. 166. Only those actually born in Europe are included in the European stock, not their children.

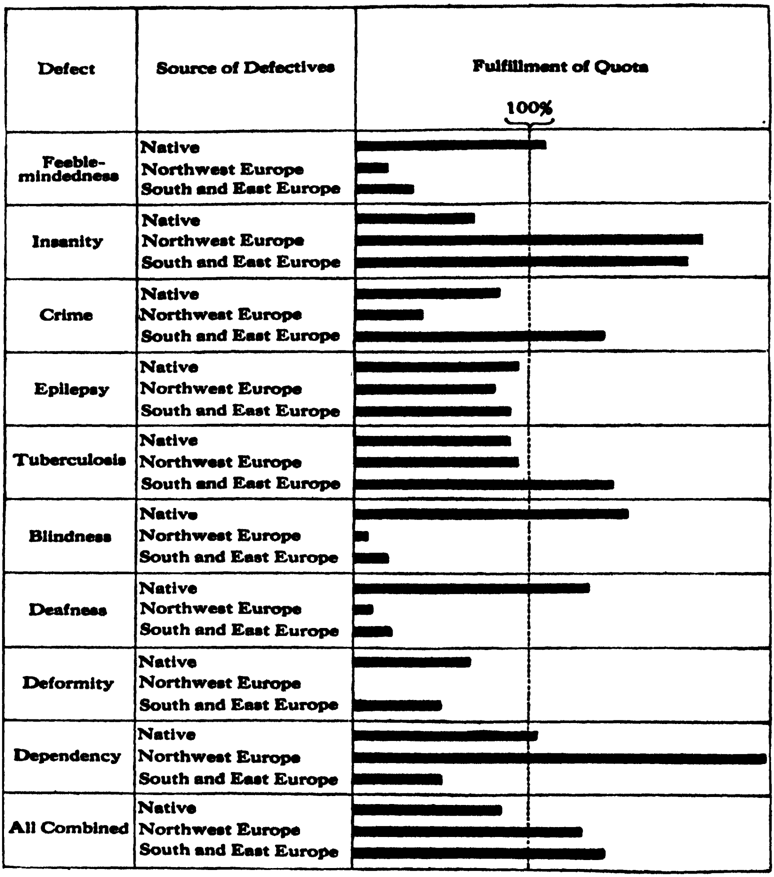


FIG. 166.—Comparison of northwestern and southeastern Europe with respect to their contribution of inmates of American custodial institutions. Quota fulfillments are indicated in the same way as in Fig. 165. Northwestern Europe shows the better record in seven respects, the worse record in two respects. Owing to the large number of the insane, one of the respects in which northwestern Europe makes a poor showing, the record as a whole is only slightly better for northwestern Europe than for the southeastern countries.

Comparison of the second and third horizontal black bands under each of the defects reveals that northwestern Europe has the advantage of southern and eastern Europe in seven cases, but is at a slight disadvantage in insanity and at a very great disadvantage

in dependency. Taking all defects together, and weighting them according to the number of individuals exhibiting them, northwestern Europe has a slightly better record than southern and

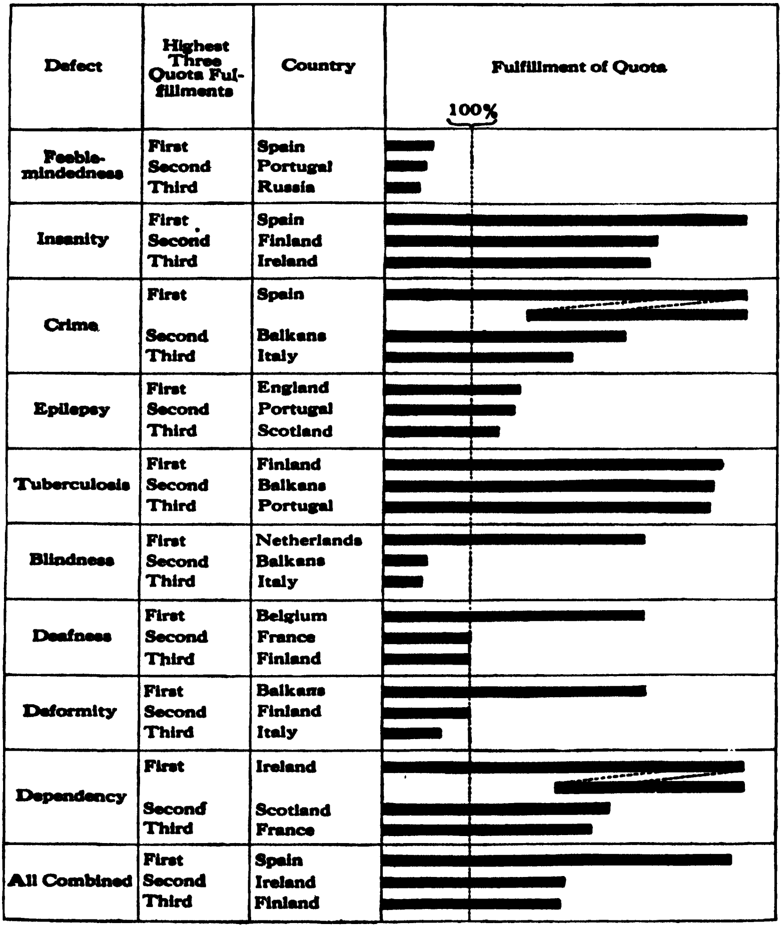


FIG. 167.—Highest three proportionate national contributions of inmates of custodial institutions for each of nine defects. The Balkan states are combined. Finland appears five times in this chart; Spain and the Balkans four times each; Ireland. Italy and Portugal each three times. Countries of south and east Europe appear twice as often as do those of northwest Europe.

eastern Europe (bottom of figure). One could wish that the children of the immigrants were included, since in this way one might arrive at an estimate of the various nationalities with respect to recesssive inherited characters, like feeble-mindedness

and epilepsy, which can to some extent or even largely be detected in those immigrants who are homozygous and can be thus excluded at the port of entry. Other obvious defects, like blindness, deafness, and deformity, could also be more correctly judged, so far as they may be due to heredity, if the children were included. The data are not published in such a way, however, that the children of immigrants may be studied.

Another comparison that is of interest is that of nations with one another. Because of the large number of these nations in Europe, this comparison is made by selecting only the three nations which supply the greatest percentage of their quotas to our institutions with respect to each defect. The Balkan states are combined, owing to the small number of immigrants from most of them, and because their quota fulfillments tend to be high or low in unison. Figure 167 gives the results. Northwestern countries appear ten times in this table, southern and eastern countries twenty times. Since the southern and eastern states, as arranged for this table, are not quite so numerous as the northwestern nations, this comparison is even more to the advantage of northwest Europe. Finland appears in the chart five times, Spain and the Balkans each four times; these are all in the south or east. But Ireland, of the northwest, is included three times, as are Italy and Portugal of the south.

Possible Misinterpretation.—The principal source of error in the conclusions to be drawn from Laughlin's study, other than those already pointed out, is the fact that it dealt only with inmates of institutions. There are many people of both immigrant and native stocks suffering from the same defects who are still at large. If the institutional defectives are truly representative of the whole group, in and out of the institutions, no serious mistakes would be made by basing policy upon them. If something, national custom, for example, should prevent the people from one country from entering institutions as freely as do those of another nation, the relative numbers in institutions would give an entirely erroneous notion of the relative desirability of those two countries as sources of immigration. It is difficult to believe, for example, that the Irish actually furnish more than twice as many dependents as any other nation, and six times their quota. One wonders whether the dependents of other nationalities may not be quartered, in as great numbers, among

their relatives, perhaps with the feeling that it is a family disgrace to let them become public charges. There is no way of knowing how great an error creeps into the conclusions from this source, short of taking a census of all defectives at large. The latter remedy can hardly be applied because of the labor required, but in its absence it is necessary to be cautious in interpretation.

Another source of error may lie in the age distribution of inmates, with respect to the mental disorders lumped together as insanity. Malzberg (1936) holds that in New York state the foreign-born are probably no more subject to this type of defect than are the natives; the foreign-born are merely older, and are concentrated in cities. Some forms of insanity do not come to expression until middle life, and there is a greater tendency to mental ills—or a greater likelihood that the unfortunate will be hospitalized and so counted—in cities than in rural districts. The age distribution of defectives in institutions and in the native and foreign-born population would have to be known to remove this source of error.

Improvement of Immigration Policy.—Probably the most useful change in the immigration policy of the United States, if it were feasible, would be to base admission on genetic constitution rather than nationality. The best people should be obtained, no matter whence they come. To determine genetic constitution, it would be necessary to examine the family histories of applicants in their home countries, before they set sail. Examinations are now given abroad to a considerable portion of the applicants, but they bear mostly if not exclusively on the qualities of the applicant himself, not his family. All persons who on this examination show any mental defect are excluded, and they constitute 1 to 3 per cent of the total, at different ports. About 9 per cent show some defect, but not all of these are rejected. Whether suitable family histories could be obtained, even by placing the responsibility of furnishing it on the applicant, may be questioned. If it proved practicable to ascertain genetic constitution from pedigrees, national allotments could well be discarded. A quota could still be set to govern total admissions.

Other changes, generally designed to make regulation more strict, have been proposed by students of immigration. The following are taken from Laughlin (1934) in a study made for

the Chamber of Commerce of the State of New York. He calls for the immediate extension of immigration restrictions to all nations, which means adding the countries of North and South America, which are now exempt. The advantage of this restriction would be chiefly the virtual exclusion of Mexicans. Mexico is the only American country now sending us large numbers. Its peon class flocks over the border in good times and goes back in bad. Racially they are quite distinct from the people of the United States, and they have caused considerable economic disturbance in the southwestern states.

To make restrictions against North American countries effective, it would be necessary either to enlarge the border patrol very greatly, or to require registration of all aliens. The latter step will probably be necessary anyway. Aliens not able to show registration certificates would then be deportable no matter where found.

Laughlin further urges that the basis of quotas be changed to the "white racial stocks" now in this country as derived from the several countries. Nations which have sent us partly or exclusively nonwhites would thus have their quotas reduced.

Other suggestions are (1) that naturalization requirements, which are now determined by local authorities, be made uniform and more strict, and (2) that for any naturalized citizen convicted of a crime which would call for his deportation if unnaturalized, citizenship be automatically canceled.

APPENDIX

QUANTITATIVE CHARACTERS

Many of the characters of organisms exist in such sharply defined forms that names suffice to distinguish them. Eye color may be scarlet, blood, or peach; plant stems may be tall or dwarf with no doubtful intermediates; feathers may be solid black or barred. It is with qualities like these that this book has chiefly dealt. They are sometimes called qualitative characters.

Often, however, the expression of a character in individuals is highly variable. Two strains of wheat have grains of unequal size, and when they are kept separate it is easy to see that one is larger than the other. Yet the individual grains are of various sizes, and the smaller grains of the large strain are smaller than the larger grains of the small strain. Such qualities may be called quantitative characters. Names for them will not suffice, they must be measured.

The Mean.—The first measure of a quantitative character is the *mean* or average of its expression in a considerable number of individuals. It is assumed that the student is familiar with ordinary arithmetical methods of calculating the mean, but the short method used in statistical work may not be so well known. The method is here illustrated with the weight of hens' eggs, whose mean is worth ascertaining in instances where two breeds differ in egg size. To simplify the calculation, it will be assumed that the eggs were weighed with the idea of the statistical study in mind, and that they have been grouped into classes. It was decided, we may suppose, to group the eggs together to the nearest tenth of an ounce; one class would include all eggs weighing between 1.85 ounces and 1.95 ounces, another class all between 1.95 ounces and 2.05 ounces. The complete data obtained are shown in Table 1, in which only the first three columns were filled.

This table shows that there were 2 eggs (frequency, f) between 1.45 and 1.55 ounces in weight (class range), 5 eggs between 1.55 and 1.65 ounces, and so on. Each class is treated as if the eggs in it were concentrated at its middle point. The third class is made up of 17 eggs weighing 1.7 ounces (class value, v), the fifth class of 60 eggs each weighing 1.9 ounces. Now, it is obvious that the mean weight of these eggs is approximately 2 ounces, and this value may be called the assumed mean (a). The mean assumed is always one of the class values. All other classes deviate from this assumed mean, in either a positive or a negative direction, and the deviation (d') is measured with the class interval as a unit. The deviation of the first class of eggs from the assumed mean is -5 class intervals; that of the eighth class is $+2$ class intervals.

Each frequency is now multiplied by the deviation of its class from the assumed mean, and the respective (negative and positive) products (fd')

TABLE 1

Class range	Class value v	Frequency f	d' −	d' +	fd' −	fd' +	fd'^2
1 45–1 55	1 5	2	5		10		50
1.55–1.65	1.6	5	4		20		80
1.65–1.75	1 7	17	3		51		153
1.75–1.85	1 8	31	2		62		124
1.85–1 95	1 9	60	1		60		60
1.95–2.05	2 0	74	0	0	0	0	0
2.05–2 15	2 1	58		1		58	58
2.15–2 25	2 2	29		2		58	116
2.25–2.35	2 3	10		3		30	90
2.35–2.45	2.4	2		4		8	32
2.45–2 55	2.5	3		5		15	75
i = 0.1		n = 291			203	169	$\Sigma fd'^2$ = 838
			$\Sigma fd'$ =		34		
$\Sigma fd'/n$ = −0.116			$\Sigma fd'^2/n$ = 2 8797				

are set down in the sixth and seventh columns. These products are summed up at the bottom, where it appears that the negative ones outweigh the positive ones, so that the net sum of the products ($\Sigma fd'$) is −34. From these numbers the mean is calculated from the formula

$$M = a + i\frac{\Sigma fd'}{n}$$

in which i is the class interval (0.1 ounce), n is the total number of eggs (291), Σ indicates summation, and the other symbols have the significance already indicated. Substituting the actual values in the formula, we have

$$M = 2.0 + (0.1)\frac{-34}{291} = 2.0 - 0.0116 = 1.9884 \text{ ounces}$$

This value of the mean may be rounded off at 1.99 ounces for any further calculations.

Significance of the Mean.—How much the mean tells regarding a population depends on how nearly it describes the individuals composing the group. When the mean weight of American silver dollars is given as so many grains, that mean is highly descriptive because every dollar weighs very close to the stated amount. Coins deviating much from the standard weight are rejected at the mint. When, however, the mean per capita wealth of a certain eastern city is given as so many dollars, that information is of little descriptive value because almost no one in the city has that wealth. For the city is made up of several multimillionaires and a considerable group of merchants and laborers. The silver dollars constitute a very

uniform population, the people of the eastern city a highly variable one. The mean of a nearly uniform group is always more informative than the mean of a very variable one.

It is necessary, therefore, to have a measure of the variability of a population in order to judge the significance of its mean. Besides, the variability itself is sometimes of considerable importance entirely apart from its relation to the mean.

Variability.—The measure of variability is the *standard deviation* (σ), defined by the formula

$$\sigma = \sqrt{\frac{\Sigma f d^2}{n}}$$

in which f is the frequency as already used in the preceding section, d is the deviation of each class from the mean, Σ is the sign of summation, and n as before is the total number of individuals. This formula is difficult to use because, since the mean nearly always ends in a fraction, d likewise involves fractions, and squaring fractions is laborious. For calculation, therefore, the following formula is substituted:

$$\sigma = i\sqrt{\frac{\Sigma f d'^2}{n} - \left(\frac{\Sigma f d'}{n}\right)^2}$$

In this formula d' is the deviation of each class, not from the actual mean, but from an assumed mean; and to save labor the mean assumed should be the same (a) as was used in calculating the mean. The deviation is thus always a whole number. It is measured in class intervals and is therefore always small, so that squaring it is an easy operation. The other symbols in the formula have the same significance as was indicated in connection with the mean.

Using the assumed mean rather than the actual mean from which to measure deviations introduces an error which is approximately corrected by deducting $(\Sigma f d'/n)^2$ before the square root is extracted.

In calculating the standard deviation from this formula, much labor is saved by preserving the calculation of the mean, as in Table 1. The values fd'^2 are readily computed (eighth column) by multiplying the deviation (d') in column 4 or 5 by the product fd' in column 6 or 7. The sum of these products is $\Sigma f d'^2$, and in this population it is 838. The quotient $\Sigma f d'/n$ was found, in the calculation of the mean, to be -0.116. We may, therefore, substitute concrete values for the symbols in the equation of the standard deviation, thus:

$$\sigma = 0.1\sqrt{{}^{838}\!/_{291} - 0.116^2} = 0.1\sqrt{2.8663} = 0.17$$

The standard deviation of these eggs is thus 0.17 ounce. The correction factor $(\Sigma f d'/n)^2$ is almost negligible in this particular population because the assumed mean ($a = 2.0$) was so near the actual mean, 1.99. In other populations when the mean is not so correctly guessed or does not come so near one of the class values, the correction factor is important.

Abstract Measure of Variability.—The standard deviation, 0.17 ounce, measures the variability of the eggs satisfactorily in all relations arising within this particular population. It does not, however, suffice to compare the variability of the eggs with the variability of the height of men in inches. It cannot be used to compare variability measured in any other unit than ounces. It cannot be used even to compare the variability of one group of eggs with that of another group of eggs, unless both lots have the same mean weight, for, if the second population of eggs has a higher mean weight, it should have also a larger standard deviation in order to have the same variability as the first group.

For comparisons of variability in different populations, the measure of variability must be changed to the *coefficient of variation,* which is defined as

$$V = \frac{\sigma}{M} = \frac{100\sigma}{M} \text{ per cent}$$

in which σ is the standard deviation, M the mean. For the population of eggs in Table 1,

$$V = \frac{0.17}{1.99} = 0.085$$

or 8.5 per cent if it is desired to express the coefficient as a percentage. This value 0.085 is an abstract number, not ounces, not inches; it is comparable with the coefficient of variation of any other population.

Direct Use of Measures of Variability.—The coefficient of variation is directly used in genetic work in deciding the degree of variability of F_1 and F_2 generations. Blending inheritance (page 153) is characterized by fairly uniform F_1 (if the parents are homozygous and all of the same genotype within each parent group) and a variable F_2. Suppose that, in an experiment testing the inheritance of the number of rows of grains in the ears of corn, two strains differing in that number have been crossed and that both F_1 and F_2 generations have been obtained, with the result shown in Table 2.

TABLE 2

Generation	Number of ears having the following rows of grains					
	6	8	10	12	14	16
Parent 1	5	7				
Parent 2	..	..	..	1	5	2
F_1	..	6	62	35		
F_2	1	12	58	29	19	2

For the F_1 generation $M = 10.56$ rows, $\sigma = 1.13$ rows, and $V = 10.7$ per cent. For the F_2 generation $M = 10.98$ rows, $\sigma = 1.90$ rows, and $V = 17.3$

per cent. The student is encouraged to verify these figures by his own computation. The contrast of 17.3 with 10.7 shows that F_2 is more variable than F_1 (which was in this instance obvious from mere inspection of the table) and that therefore this particular condition of blending inheritance is met. The number of rows of grains is presumably a multiple-gene character.

Normal Curve of Variation.—Turning now to the use of variability in judging of the value of the mean, we must first observe the normal distribution of the values of a variable character. If any measurable quality fluctuates (in a large number of individuals) in a purely random manner about a mean condition, a graph of the values of the character in the whole population tends to take the form of the curve in Fig. 168,

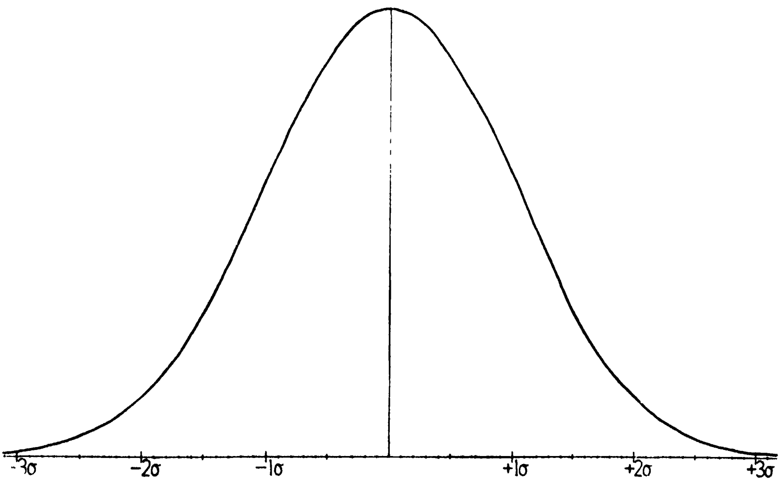


FIG. 168.—Normal curve of variation, with multiples of the standard deviation marked on the base line.

which may be called the normal curve of variation. It rises to a peak in the middle, just as the weights of eggs in Table 1 would do if they were plotted, and falls off symmetrically to right and left, almost to zero.

This curve has certain properties in relation to the standard deviation. If distance be marked off on the base line (with the standard deviation as the unit) in plus and minus directions from the mid-point which represents the mean, it is found that practically the entire population lies between two extremes, one of which is three times the standard deviation above the mean, the other three times the standard deviation below the mean. If the mean of a population is 20 and its standard deviation is 2, very few of the individuals will be over 26 or under 14, that is, if the population is distributed in the normal random manner. Moreover, between these extremes, certain fractions of the population lie at certain distances from the mean. These fractions and their respective distances (measured with the standard deviation as the unit) are given in part in Table 3.

This table shows, for example, that between the mean and 0.35 times the standard deviation above or below the mean (see seventh line of table) there is 0.1368 of the whole population; between the mean and 0.90 times the standard deviation above or below the mean is 0.3159 of the population; and between the mean and 2.10 times the standard deviation above or below the mean is 48.21 per cent of the population. The table may be

TABLE 3

$M - x\sigma$	Fraction of total population	$M - x\sigma$	Fraction of total population	$M - x\sigma$	Fraction of total population
0.05	0.0199	1.35	0.4115	2.65	0.4960
0.10	0.0398	1.40	0.4192	2.70	0.4965
0.15	0.0596	1.45	0.4265	2.75	0.4970
0.20	0.0793	1.50	0.4332	2.80	0.4974
0.25	0.0987	1.55	0.4394	2.85	0.4978
0.30	0.1179	1.60	0.4452	2.90	0.4981
0.35	0.1368	1.65	0.4505	2.95	0.4984
0.40	0.1554	1.70	0.4554	3.00	0.4987
0.45	0.1736	1.75	0.4599	3.05	0.4989
0.50	0.1915	1.80	0.4641	3.10	0.4990
0.55	0.2088	1.85	0.4678	3.15	0.4992
0.60	0.2257	1.90	0.4713	3.20	0.4993
0.65	0.2422	1.95	0.4744	3.25	0.4994
0.70	0.2580	2.00	0.4772	3.30	0.4995
0.75	0.2734	2.05	0.4798	3.35	0.4996
0.80	0.2881	2.10	0.4821	3.40	0.4997
0.85	0.3023	2.15	0.4842	3.45	0.4997
0.90	0.3159	2.20	0.4861	3.50	0.4998
0.95	0.3289	2.25	0.4878	3.55	0.4998
1.00	0.3413	2.30	0.4893	3.60	0.4998
1.05	0.3531	2.35	0.4906	3.65	0.4999
1.10	0.3643	2.40	0.4918	3.70	0.4999
1.15	0.3749	2.45	0.4929	3.75	0.4999
1.20	0.3849	2.50	0.4938	3.80	0.4999
1.25	0.3944	2.55	0.4946	3.85	0.4999
1.30	0.4032	2.60	0.4953	3.90	0.5000

used backwards. If one wants to know within what limits, equidistant from the mean on either side, 80 per cent of the population is to be found, it is only necessary to look at the bottom of the first column where the limit of approximately 40 per cent (0.4032) is given. It appears there that 80.64 per cent lies within the limits of 1.30σ above and 1.30σ below the mean. If more precise limits are to be set, a more complete table is needed, or one must interpolate between the points given.

This table of distribution of a normal population is most important in judging of the significance of means and other statistical values.

Reliability of the Mean.—The means with which statistical work deals are usually computed from only a small fraction of the individuals which might have been included. If the mean thus obtained is near the mean of the whole group, it is reliable; if the mean of a limited number is apt to be far from the mean of the whole existent population, it is unreliable. Reliability depends on how variable the population is, and on how many individuals have been obtained from it for study and measurement. How a measure of reliability is to be devised should be clear from the following considerations.

If an investigator today takes 500 individuals from a population consisting of a million or more, he obtains from them a certain mean. Tomorrow he takes another 500 and obtains another mean It is not the mean obtained from the first 500, and neither mean is that of the entire million. He and other investigators take various samples, not always the same number of individuals, and calculate from them various means. Probably no mean is identical with any other one. Yet they tend to cluster about a certain value. Indeed, the distribution of these means is of the same sort as the distribution of individuals in Fig. 168. The means all fall within a certain range. The bulk of them accumulate near the middle of that range, while fewer and fewer are successively farther and farther from the middle point. Theoretically the value around which the various means tend to collect is the real mean of the population of a million, or whatever the total number is. In statistical work one is constantly trying to judge what that real mean is from the mean of a much smaller number of individuals.

Standard Error of the Mean.—These attempts focus on the calculation of what is called the standard error of the mean This standard error may be symbolized by σ_M, and is defined by the equation

$$\sigma_M = \frac{\sigma}{\sqrt{n}}$$

in which σ is the standard deviation of the limited group whose mean is being judged as to reliability, and n is the number of individuals from which the mean was computed. The letter σ is used as the symbol of the standard error because that error corresponds to the standard deviation of a large number of means separately calculated from different samples of the entire extant population. Consequently the standard error may be used as the unit of measurement in Table 3, in place of the standard deviation Some practice in making this use of Table 3 is desirable.

To make the judgment concrete, we may return to the weights of hens' eggs in Table 1. The mean weight of these eggs was 1.99 ounces, their standard deviation was 0.17 ounce, and the number of eggs was 291 Substituting the pertinent numbers in the equation of the standard error of the mean, we find that

$$\sigma_M = \frac{\sigma}{\sqrt{n}} = \frac{0.17}{\sqrt{291}} = 0.01 \text{ ounce}$$

This standard error of the mean, 0.01 ounce, is indicated by writing the mean as 1.99 ± 0.01 ounces.

What does this standard error tell us? Since the standard error corresponds to a standard deviation, Table 3 is the criterion of judgment. Three times the standard error above and below the computed mean are limits within which the real mean is almost certain to lie. These limits for the hens' eggs are 2.02 and 1.96 ounces, respectively. Though the mean of only 291 eggs has been ascertained, it is practically certain that, had an indefinitely large number of eggs from the same source been weighed, their mean would not have been over 2.02 or under 1.96 ounces. If a narrower limit be set, say two times the standard error, it is found from the center of Table 3 that 95.44 per cent (twice 0.4772) of the chances are that the real mean weight is within this narrower range. That is, if all the possible eggs from that source had been weighed, there are less than 5 chances in 100 that their mean would have been more than 2.01 or less than 1.97 ounces. Still narrower limits may be set, and Table 3 shows what chance there is that the real mean falls within them.

Most investigators are content to regard 9974 chances out of 10,000 as practical certainty (see eighth line of last columns of Table 3), and consider that the real mean of any indefinitely large population is not more than three times the standard error away from the computed mean of a limited portion of that population.

Other Standard Errors.—Every other value computed in statistical work has its standard error, which is the measure of reliability. Everywhere it corresponds to the standard deviation, and Table 3 is used to determine the significance of the quantity to which it relates. For the standard deviation the standard error is

$$\sigma_\sigma = \frac{\sigma}{\sqrt{2n}} = 0.7071\sigma_M$$

For the coefficient of variability the standard error is

$$\sigma_v = \frac{V}{\sqrt{2n}}\sqrt{1 + 2\left(\frac{V}{100}\right)^2}$$

One of the most useful of standard errors in genetic work is that pertaining to a proportion. It is desired to know how far the actual ratio obtained in an F_2 generation may depart from the expected 3:1 without indicating differential mortality, lethal homozygosis, or indeed anything else than chance. The standard error of the ratio gives that information. If the group under consideration is expressed as a fraction of the whole, the formula of the standard error is

$$\sigma_p = \sqrt{\frac{p(1-p)}{n}}$$

in which p is the proportion, and n is the total number of individuals. Thus, in an F_2 generation consisting of 289 individuals of which 205 are of one kind, 84 of another, how likely is it that this distribution differs in any but an

accidental way from a ratio of 3:1? The actual proportion in the majority group is 0.7093. The standard error of this proportion is accordingly

$$\sigma_p = \sqrt{\frac{0.7093 \times 0.2907}{289}} = 0.0267$$

It requires only 1.52 times this standard error to be added to 0.7093 to make it the theoretical 0.75, so according to Table 3 there are about 13 chances in 100 that the drop to 0.7093 is purely accidental.

Without converting the numbers of individuals into a proportion, the standard error can be computed according to the formula

$$\sigma = \sqrt{\frac{c(n - c)}{n}}$$

in which c is the number of members of one of the two classes, and n is the total number of individuals. The standard error thus computed is in individuals, not a fraction. Its significance and use in connection with Table 3 are not in any way changed. The major class of the pair just used is

$$205 \pm \sqrt{\frac{205 \times 84}{289}} = 205 \pm 7.7$$

It would take adding only 1.52 times this standard error to 205 to make it the 216.75 that would be exactly three-fourths of the total.

Standard Error of a Difference.—Frequently it is desired to know whether one population differs significantly from another as indicated by their means or whether one is more variable than another as indicated by their standard deviations. Each mean or each standard deviation has been calculated with its own standard error. The difference between the means, for example, is found by simple subtraction. The standard error of that difference is determined from the errors of the two means by the equation

$$\sigma_d = \sqrt{\sigma_a^2 + \sigma_b^2}$$

in which σ_a is the standard error of the one mean (or other measure) and σ_b is the standard error of the other mean. If one mean is 28.6 ± 0.93 and the other is 26.9 ± 0.54, the difference between them is

$$1.7 \pm \sqrt{(0.93)^2 + (0.54)^2} = 1.7 \pm 1.07.$$

The difference is only 1.59 times its own standard error, and, by referring to Table 3, we find that there are about 89 chances in 100 that the two means represent really different populations, or 11 chances in 100 that the difference is due only to the accidents of random sampling. A difference must be at least three times its standard error to make it practically certain (9974 chances in 10,000) that it is not merely accidental.

Probable Error.—Some investigators prefer to use a measure of reliability which divides the chances of significance and insignificance evenly. They wish to attach to any uncertain measure an error which is just as likely to

be exceeded as not. To ascertain what this error is it is only necessary to find in Table 3 that multiple of the standard deviation which marks off 0.25 of the population (which is 0.50 when fluctuation both positively and negatively is included). This multiple is nearly midway between 0.65 and 0.70 times the standard deviation. By interpolation it is found to be 0.6745. Hence, an error which is 0.6745 times the standard error is chosen. This measure of reliability is called the probable error. The probable error of a mean is

$$\text{P.E.}_M = 0.6745\sigma_M = \frac{0.6745\sigma}{\sqrt{n}}$$

The probable error of a standard deviation is

$$\text{P.E.}_\sigma = 0.6745\sigma_\sigma = \frac{0.6745\sigma}{\sqrt{2n}}$$

Any probable error may be found by multiplying the corresponding standard error by 0.6745.

There is no advantage in the probable error, but it is still frequently used. It entails more labor: (1) it requires one more multiplication to calculate it; and (2) if Table 3 is to be used in forming judgments of probability, the probable error must first be divided by 0.6745 before the table is of service.

In connection with error, either standard or probable, it should be made clear that it does not refer to any mistake in calculation or in the original observations or measurements. It is merely the error which one makes in assuming that the entire existent population is like the limited sample which has been studied.

Correlation.—Many geneticists find the principle of correlation useful as evidence of heredity when other signs fail. Correlation is a connection between two properties of the individuals of a population such that, as one of the properties varies, the other tends to vary. If as one property increases the other tends to increase, the correlation is positive; if as the one property increases the other tends to decrease, the correlation is negative. Positive correlation indicates that the two qualities have part of their physiological bases in common, and the physiological bases are not infrequently genetic.

The most commonly used measure of correlation is Pearson's coefficient of correlation, defined by the equation

$$r_{xy} = \frac{\Sigma d_x\, d_y}{n\sigma_x\, \sigma_y}$$

but calculated from a more complicated equation to be given later. To use it, one must first have measures of the two qualities in each of a considerable number of individuals. The data are collected with the idea of correlation in mind and are entered in a table ruled in squares. To make the discussion concrete, suppose that one contemplates ascertaining any correlation between the depth of yellow color in the ear lobes of fowls and the number of eggs they lay. A color chart or some more objective way of determining yellow must be available, and we may arbitrarily divide the range of color into nine grades, 1 the palest, 9 the darkest yellow. If it be guessed that the

poorest layers will produce at least 100 eggs in a year, the best ones less than 200, the egg production may be divided into 10-egg classes, beginning 100–109, 110–119, and ending 190–199.

On a sheet of cross-section paper the egg production classes may be written along the top, and the color classes down the left margin. Suppose the first hen is of color grade 5 and lays 132 eggs; a dot is placed in the square in the fifth row and fourth column. For a hen which is of color grade 2 and lays 160 eggs, a dot is placed in the square in the second row and seventh column. Each fowl is recorded by a dot. When the data are complete, the dots in the several squares are counted and the number written in place of them. The result is what is called a correlation table (Table 4).

TABLE 4

No. of eggs / Color	100 to 109	110 to 119	120 to 129	130 to 139	140 to 149	150 to 159	160 to 169	170 to 179	180 to 189	190 to 199	Color
1						1 −4					1
2				4 +12			2 −12		1 12		7
3		2 +12			7 0	4 −8	2 −8	2 −12			17
4			9 +18	8 +8	14 0	8 −8	8 −16	4 −12	2 −8		53
5		7 0	6 0	13 0	11 0	9 0	7 0	4 0		1 0	58
6		3 −9	5 −10	10 −10	12 0	10 +10	5 +10				45
7	1 −8	4 −24	3 −12	5 −10	7 0	6 +12	3 +12				29
8		2 −18		1 −3	2 0	2 +6		1 +9			8
9			1 −8			1 +4					2
Eggs	1	18	24	41	53	41	27	11	3	1	220

By summing up the horizontal rows there is obtained at the right the distribution of the color classes of the hens; and by adding the columns there is placed at the bottom the distribution of the egg production of the same hens. For each of these distributions there is calculated usually the mean and always the standard deviation, by the methods already described. The calculations should be preserved, for some portions of them enter into

the computation of the coefficient of correlation. Suppose that the assumed mean color grade was 5, and the assumed mean egg production was 144.5, both being the middle points of the ranges of the most numerous classes. If these classes are marked off by heavy lines, the table is divided into quadrants.

The equation used to calculate r differs from the defining one already given, because assumed means have been used in order to save labor. This calculating formula is

$$r_{xy} = \frac{\Sigma f d_x' d_y' - \dfrac{(\Sigma f d_x')(\Sigma f d_y')}{n}}{n s_x s_y}$$

in which d_x' is the deviation of any group from the assumed mean egg production, d_y' is the deviation of the same group from the assumed mean color grade, f is the number in the respective squares of the table, n is the number of hens in the total sample, s_x is the standard deviation of egg production in class intervals (not eggs), s_y is the standard deviation of color grade in class intervals, and Σ is the sign of summation.

The first term in the numerator of the formula is now computed. The frequency 2 in the upper left quadrant of Table 4 is that of a class which deviates -3 from the assumed mean egg production and deviates -2 from the assumed mean depth of color. For this group, therefore, $fd_x'd_y'$ is $2(-3)(-2)$, which is $+12$. This product is set in small figures in the square with the frequency. For the group of 4 in the lower left quadrant, the deviation from one mean is -3, from the other $+2$; hence $fd_x'd_y'$ is $4(-3)(+2)$ or -24. The corresponding products are computed for the other groups in the table. In the assumed mean classes all products are 0 because one of the deviations is 0. In the upper left and lower right quadrants all products are positive, those of the upper right and lower left are all negative. The quantity $\Sigma fd_x'd_y'$ is the sum of all these products, with their signs taken into account; for this particular table it is -99.

The value of $\Sigma fd_x'$, used in computing the mean egg production, was found to be -2. That of fd_y', used in determining the mean color grade, was $+23$. The total number n is 220; σ_x is 17.0 eggs, hence s_x is 1.7 class intervals; and σ_y is 1.45 color grades, so that s_y is 1.45 class intervals. If these concrete values be substituted in the equation, it is found that $r = -0.18$. The correlation is negative; as egg production increases, depth of yellow color is diminished.

Values of r range from 0 to 1, either positive or negative. If one quality has no relation whatever to the other, r is 0. If, for a given value of one variable, there is inevitably a certain value of the other variable, r is 1. The relation between ear-lobe color and egg production is therefore relatively slight. The standard error of the coefficient of correlation is

$$\sigma_r = \frac{1 - r^2}{\sqrt{n}}$$

However, since r is not a very accurate measure of the common basis of the correlated properties, the standard error is of less use than elsewhere.

For Table 4, $\sigma_r = 0.065$; the coefficient of correlation is thus only 2.77 times its error, hence just possibly not significant.

Genetic Uses of Correlation.—In asexually reproducing organisms, the heredity of a variable character cannot be proved by comparison of parents and offspring in a single family, since the parents might accidentally be below the mean of their strain while the offspring might, likewise accidentally, be above the mean of their strain. Size of the protozoon Paramecium fluctuates tremendously without any genetic basis for the variability; the number of spines on the shell of Difflugia varies less, also without any corresponding genetic change. If in these examples the character of the parents be plotted against the mean of their offspring in a correlation table and a positive coefficient of correlation is obtained, this is evidence that the size or the number of spines has a genetic foundation as well as an environmental one.

In man a quantitative character like the nasal index may vary so much as to leave one in doubt, in single pedigrees, whether it is inherited or not. If in this situation a number of identical twins are available, correlation between them may be compared with correlation between fraternal twins. If the former correlation has a higher coefficient than the latter, nasal index is partly hereditary.

QUESTIONS AND PROBLEMS

The following questions of objective type and brief problems should serve as a test of accomplishment after study has been completed. They are not numerous enough, however, to form the primary basis of review. Some of the questions are not directly answered in this book; for them the student should draw on his store of general biological information. There are three types of tests: (1) true or false statement, (2) completion test, and (3) multiple choice statement. These are separated into distinct lists for simplicity of administration. Within each list the questions follow roughly the order of presentation in the text; for questions on any topic, therefore, it is necessary to turn to the pertinent parts of all three lists.

PART I

Each of the statements following is either true or false. Accordingly, write in the parenthesis before it the word "true" or "false," the sign + or −, or the word "yes" or "no." A statement must be entirely true to be recorded so. A parenthesis left blank indicates uncertainty. To ascertain the weight to be assigned to each true or false statement in an examination, divide the number of points represented by the whole group of statements by the number of statements. The quotient is the amount to be deducted from 100 for each statement not marked; twice that amount should be deducted for each mistake. For example, if this type of test is weighted 40 in the examination as a whole, and there are 30 statements, $1\frac{1}{3}$ should be deducted for each statement not marked, and $2\frac{2}{3}$ for each one marked incorrectly.

Chapter I

( ) **1.** Aristotle knew that mules are usually sterile.

( ) **2.** Historical records show when wheat was first cultivated.

( ) **3.** European academies of science were generally convinced, by the year 1800, that plants hybridize.

( ) **4.** That the various characters of the parents may recombine in different ways in their hybrid offspring was known before Mendel's experiments were performed.

( ) **5.** Mendel found that his predecessors in plant hybridization had reported accurately the numbers of each kind of individual in hybrid generations.

( ) **6.** Darwin's interest in evolution hastened the recognition of Mendel's work.

( ) **7.** Mendel's experiments influenced Charles Darwin's theories greatly.

Chapter II

( ) **8.** Somatic cells regularly produce germ cells in most higher animals.
( ) **9.** Somatic and germ cells are more sharply distinguished in plants than in animals.

Chapter III

( ) **10.** All cells of one animal have the same number of chromosomes.
( ) **11.** The chromosomes in one cell are all alike.
( ) **12.** The material at one end of a chromosome is like that at the other end.
( ) **13.** Division of the body of the cell is the most important part of cell division.
( ) **14.** A chromosome may be equally divided by cutting it crosswise in the middle of its length.
( ) **15.** Plastids owe their characteristics to chromosomes.

Chapter IV

( ) **16.** Parthenogenetic eggs undergo maturation.
( ) **17.** Asexual reproduction makes for stability of a species, as compared with sexual reproduction.
( ) **18.** Mutation occurs in asexual organisms as well as in sexual ones.
( ) **19.** The parthenogenetic eggs of one individual animal are as likely to differ among themselves if they have undergone only one maturation division as if they have undergone two.

Chapter V

( ) **20.** Somatic cells contain some genes that do not come to expression in those cells.
( ) **21.** A skin cell and nerve cell of the same animal may contain different numbers of chromosomes.
( ) **22.** Symmetry of an embryo is usually determined by the chromosomes of the egg from which it develops.
( ) **23.** Symmetry of an insect is settled before the egg from which it develops even begins to divide.
( ) **24.** Some geneticists hold that genes may mutate as a regular part of somatic differentiation.

Chapter VI

( ) **25.** Any chromosome may pair with any other chromosome in the same cell in maturation.
( ) **26.** Any paternal chromosome may pair with any maternal chromosome in the same cell in maturation.
( ) **27.** A given chromosome may pair with only a particular other chromosome in maturation.
( ) **28.** Reduction division occurs before equation in some animals, after equation in others.

( ) **29.** The mature germ cells of an animal may contain more maternal chromosomes than its body cells contain.

( ) **30.** A skin cell and nerve cell of the same animal may contain different numbers of maternal chromosomes.

( ) **31.** The maternal chromosomes in a male animal are likewise maternal in his children.

( ) **32.** Of 10 chromosomes in a mature spermatozoon, 5 are always maternal.

( ) **33.** If a primary spermatocyte contains 6 chromosomes, there are three different ways in which these 6 chromosomes may unite in pairs.

NOTE:—Whenever the question of the time of the reduction division arises in these tests and no information concerning it is given, assume that the first maturation division is reductional.

( ) **34.** Of 22 chromosomes in a secondary oocyte, 15 may be paternal.

( ) **35.** Homologous parts of two chromosomes lie opposite one another in pairing.

( ) **36.** A spermatozoon has twice as many chromosomes as a secondary spermatocyte of the same animal.

( ) **37.** Segregation of genes is brought about by reduction division of the chromosomes.

( ) **38.** All the pairs of chromosomes may have their paternal members turned toward the same end of the reduction spindle in a spermatocyte.

( ) **39.** A moss sporophyte has twice as many chromosomes as the gametophyte of the same species.

( ) **40.** The similarity of the genes in them is part of the reason for the pairing of homologous chromosomes early in maturation.

Chapter VII

( ) **41.** All of the germ cells of a roan cow may be alike.

( ) **42.** Pink flower color in snapdragons is due to a pair of identical genes.

( ) **43.** Homozygous roan cattle are more common than heterozygous roans.

Chapter VIII

( ) **44.** If an F_2 generation shows a ratio of 1:2:1, one of the genes concerned is dominant over the other.

( ) **45.** Individuals heterozygous for a dominant character are distinguishable from those homozygous for the same dominant character.

( ) **46.** Individuals heterozygous for a recessive character are distinguishable from those homozygous for the same recessive character.

( ) **47.** One of a group that makes up three-fourths of an F_2 generation exhibits the recessive character.

( ) **48.** If a heterozygote is different from both the corresponding homozygotes, it exhibits a dominant character.

( ) **49.** The ratio of 3:1 in F_2 indicates that the F_1 generation looked like one of its parents.

( ) **50.** If an animal *Aa* can be distinguished from both *AA* and *aa* by appearance, *A* is dominant over *a*.

( ) **51.** A character that appears in one or more individuals in each generation in a given line of descent is recessive.

Chapter IX

( ) **52.** If a backcross results in only one distinguishable class of offspring, the parents looked alike.

( ) **53.** If a backcross results in only one detectable class of offspring, one of the characters involved in the cross is dominant over its alternate.

( ) **54.** Crossing *Hh* × *hh* might yield 18 *Hh* and 16 *HH* offspring.

Chapter X

( ) **55.** A man receives his sex-linked characters only from his mother.

( ) **56.** A sex-linked character should appear in both sexes if many individuals exhibiting it are known.

( ) **57.** A recessive female butterfly was mated with a dominant male, and both the F_1 and the F_2 generation from them showed whether the difference between them was a sex-linked character.

( ) **58.** A gene in a W chromosome is transmitted from mother to daughter.

( ) **59.** A woman transmits her sex-linked characters only to her sons.

( ) **60.** A male fly is often homozygous for sex-linked characters.

( ) **61.** The spermatozoa of a male moth may contain the W chromosome.

( ) **62.** The genes for sex-linked characters in man are in the Z chromosome.

( ) **63.** If a male bug has 16 chromosomes, one of them is a Y.

( ) **64.** A dominant sex-linked character shows in female vinegar flies only if inherited from both parents.

( ) **65.** A recessive sex-linked mutation arising in a species of moth in nature should appear chiefly in the males.

Chapter XI

( ) **66.** If gene *A* is 13 units from one end of a chromosome, gene *a* is 26 units from one end of the homologous chromosome.

( ) **67.** A blood that contains agglutinogen A in the red cells may have agglutinin α in the plasma.

( ) **68.** Any gene at a certain locus in chromosome 1 of a certain individual is allelic to any gene at the same locus in chromosome 1 of any other individual.

Chapter XII

( ) **69.** Many homozygous Star-eyed Drosophilas have been bred.

( ) **70.** A hemophilic man receives the gene for hemophilia from both of his parents.

( ) **71.** A gene which produces a distinguishable character in heterozygotes but which is lethal at an early embryonic stage in homozygotes can be assuredly regarded as dominant over its alternate gene.

Chapter XIII

( ) **72.** An animal heterozygous for 4 pairs of genes produces 32 kinds of germ cells.

( ) **73.** Crossing $AaBb \times AaBb$ produces more different kinds of offspring than crossing $AaBb \times aabb$, provided A and B are both dominant over their alleles.

( ) **74.** Polled red Shorthorn cows crossed with horned white bulls produce all polled roan offspring. If two of these F_1 animals are bred together, the chance that their first calf will be polled white is 3 in 16.

( ) **75.** Vermilion eye is sex-linked and recessive to the wild-type red in Drosophila; sooty body is autosomal and recessive to the wild-type gray. If a red-eyed sooty female is crossed with a vermilion gray and the F_1 flies are mated together, all the vermilion sooty and vermilion gray members of F_2 will be males.

( ) **76.** Four-ninths of the dark agouti Angora rabbits in the F_2 generation listed on page 139 have the genotype *GgDdll*.

( ) **77.** Two animals differing in 4 independent pairs of genes, if crossed and their offspring are mated, yield an F_2 generation of 243 different genotypes.

Chapter XIV

( ) **78.** Blending characters are often due to multiple genes.

( ) **79.** If black is recessive in the female and dominant in the male in Ayrshire cattle, a black Ayrshire cow mated with a red bull may produce a black female calf.

( ) **80.** A human character may be dominant in one general family relationship, recessive in another.

( ) **81.** The irregular dominance of polydactyly may be explained by assuming that the polydactyl genes are different in different individuals.

( ) **82.** Dominant white in animals is commonly due to an inhibiting gene.

( ) **83.** Recessive white in animals is frequently due to lack of pigment genes.

Chapter XV

( ) **84.** If black-chaffed oats *BbGg* is crossed with gray *bbGg*, three-eighths of the offspring should be gray.

( ) **85.** If a White Leghorn hen *Iicc* is mated with a colored cock *iiCc*, half of their offspring should be colored.

( ) **86.** A species which has arisen from another species by duplication of the latter's chromosomes should have just two genes at any given locus in each cell.

( ) **87.** In sweet peas *ccRrBB* is phenotypically like *CCrrbb*.

Chapter XVI

( ) **88.** All teasel stems twist if the plants are well fed.

( ) **89.** A character which is determined by hormones may still be inherited.

( ) **90.** Hormones artificially injected into an animal act in the same way as the corresponding hormones secreted in the body.

( ) **91.** If a teasel plant produces only straight-stemmed offspring when these are well nourished, it will produce only straight-stemmed plants under any conditions.

( ) **92.** A primula whose first flowers are red may open white flowers later.

( ) **93.** Transplanting a teasel plant several times may keep its stem straight.

( ) **94.** Nutrition modifies the color of some moths.

( ) **95.** Some characters generally regarded as racial in man are modified by endocrine secretions.

( ) **96.** A character that is modified by environment is thereby excluded from being inherited.

Chapter XVII

( ) **97.** A die is as likely to fall on its 2-spot as on its 6-spot.

( ) **98.** A die that falls twice as often on its 5-face as on its 2-face is loaded.

( ) **99.** All chromosome pairs in the fly Sciara are turned either way purely by accident on the reduction spindle.

( ) **100.** When placement of chromosome pairs on the reduction spindle is purely random, each combination of chromosomes in germ cells is as likely to be produced as any other combination.

( ) **101.** If a male producing two kinds of sperm is mated with a female producing four kinds of eggs, they should produce six kinds of offspring, if the kinds of eggs do not duplicate any of the kinds of spermatozoa.

( ) **102.** It may be observed in microscopic preparations that chromosome pairs are turned at random, one way or the other, on the reduction spindle.

( ) **103.** If a die falls on its 3-spot six times, and on its 4-spot forty-one times, in 150 throws, its center of gravity is probably not in the center of the cube.

( ) **104.** Three pairs of chromosomes may be arranged in 12 ways on the reduction spindle.

( ) **105.** There are some animals in which pairs of chromosomes are not placed at random on the reduction spindle.

( ) **106.** If all cells that are normally functional survive, a female with the formula *Aa* may produce 63 eggs with the formula *A* and 66 eggs of formula *a*.

( ) **107.** If all cells that are normally functional survive, a male with the formula *Cc* may produce 1,467,286 spermatozoa containing the gene *C* and 1,467,317 with the gene *c*.

( ) **108.** Six pairs of chromosomes may be arranged in 128 ways on the spindle of the reduction division.

( ) **109.** Four pairs of chromosomes can be arranged on the reduction spindle in twice as many ways as three pairs can.

( ) **110.** According to Mendel's laws the children of the same parents should usually differ from one another.

Chapter XVIII

( ) **111.** If *CcDdEe* × *ccddee* produce 1000 offspring, of which one class appearing CDe includes 351 individuals, the genes *C*, *D*, and *E* are all in different chromosome pairs.

( ) **112.** If an animal whose genotype is *RrSsTt* produces 1020 eggs of which 127 are *rSt*, 121 *rST*, and 130*RSt*, the three pairs of genes are independent of one another.

( ) **113.** Crossing over can be detected in a mating of *AAbb* with *aabb*.

( ) **114.** When two genes get together in a chromosome as a result of crossing over from different chromosomes, they are more likely to separate again the next generation than to stay together.

( ) **115.** A male Drosophila whose genotype is *St-sT* produces spermatozoa whose formulas are *ST* and *st*, among others.

( ) **116.** Crossing over can be detected in an animal whose genotype is *Gh-Gh*.

( ) **117.** Crossing over probably occurs in an animal whose genotype is *aB-aB*.

( ) **118.** Crossing over occurs in the male of most species.

( ) **119.** Crossing over occurs as often in the male as in the female in most species in which it occurs in both sexes at all.

( ) **120.** There are more groups of linked characters in Drosophila than there are pairs of chromosomes.

( ) **121.** No organism is yet known to have more groups of linked characters than it has pairs of chromosomes.

( ) **122.** A red-eyed gray-bodied male Drosophila, heterozygous for pink eye and ebony body, mated with a pink ebony female, produces 78 pink-gray, 84 red-ebony, 16 pink-ebony, and 14 red-gray offspring.

( ) **123.** If *AB-ab* × *aabb* produce, among a total of 120 offspring, 50 of one kind, and *Cd-cD* × *ccdd* produce 60 of one kind in a total of 180, *A* and *B* are farther apart in the chromosome than *C* and *d* are.

( ) **124.** A male Drosophila whose genotype is *MS-ms*, mated with *mmss*, produces 64 offspring of one kind in a total of 178.

( ) **125.** *AaBb* may produce 864 germ cells *AB*, 872 *Ab*, 216 *aB*, 209 *ab*.

( ) **126.** If the offspring of *RrTt* × *rrtt* are 28 *RrTt*, 26 *Rrtt*, 30 *rrTt*, and 27*rrtt*, the genotype of the first-named parent was (*RT*)(*rt*).

( ) **127.** *AB-ab* × *aabb* might easily yield offspring of four kinds in the following percentages: 48, 30, 5, 17.

( ) **128.** An organism with 20 chromosomes in its somatic cells may have its hereditary characters linked in 12 groups.

( ) **129.** There is a 50 per cent chance that two human characters named at random are linked.

Chapter XIX

( ) **130.** The fact that the chromosomes in maturation behave as the genes must behave is evidence that the genes are in the chromosomes.

( ) **131.** Gametophytes derived from a heterozygous moss sporophyte are of two kinds with respect to each heterozygous pair of characters.

( ) **132.** It is known from the life cycle of a moss that segregation of genes and the reduction division of chromosomes occur about the same time.

( ) **133.** A normal moss gametophyte may have two similar genes for shape of leaf.

( ) **134.** A moss sporophyte contains genes for characters which it can never exhibit.

( ) **135.** Any part of the moss cycle which is diploid has two genes of each pair.

( ) **136.** The zygote of an alga has two genes of each pair.

Chapter XX

( ) **137.** Sex-linked characters are Mendelian.

( ) **138.** Variegation in plants is usually determined by genes in chromosomes.

( ) **139.** Grains of corn from a green plant produce striped plants.

( ) **140.** The offspring from two reciprocal crosses are alike in instances of maternal inheritance.

( ) **141.** A silkworm moth egg may be green and yet contain only the gene for white shell.

( ) **142.** What the direction of coiling of the shell will be in a snail is determined in the egg from which it develops before the egg is fertilized.

( ) **143.** A mammalian mother has more influence on her offspring than the father has.

Chapter XXI

( ) **144.** A litter of armadillos consists usually of two males and two females.

( ) **145.** Broods of insect multiple embryos sometimes include both sexes.

( ) **146.** Assuming the males in mixed broods of multiple embryos to be due to nondisjunction of the X chromosome in part of the embryonic mass is a satisfactory explanation of them.

( ) **147.** Mammals castrated when young develop secondary sexual characters as well as do normal animals.
( ) **148.** Fowls looking like cocks sometimes lay eggs.
( ) **149.** Grafting a piece of skin from one fowl to another changes the genes in it.
( ) **150.** The fact that multiple embryos are all of the same sex proves that sex is determined as early as fertilization of the egg
( ) **151.** Bonellia is potentially hermaphroditic in the larval stage.
( ) **152.** At least one fowl, after functioning as a female, has been converted into a functional male.
( ) **153.** The proportion of males can be artificially increased or diminished in rotifers.
( ) **154.** Sex in man can be controlled at will.
( ) **155.** There is but one sex gene in the X chromosome in Drosophila.
( ) **156.** A freemartin in cattle is a female of arrested or reversed sex development.
( ) **157.** Changing the sex ratio is the same as determining sex.

Chapter XXII

( ) **158.** One species of Oenothera arose from another by doubling its number of chromosomes.
( ) **159.** When a mutation arises, it generally occurs in only one individual among many in the same environment.
( ) **160.** Any agent which can reach the germ cells directly has a conceivable chance of producing mutations.
( ) **161.** Genes may produce different characters merely by being moved to some other place in the chromosomes.
( ) **162.** Interfertility of species is usually as great as fertility within species.
( ) **163.** Identical mutations have arisen in different species independently
( ) **164.** Species whose germ cells mature at different seasons should be connected by many intermediate individuals bridging over the gap between their characters.

Chapter XXIII

( ) **165.** Stiff fingers, due to fusion, without shortening, of bones at joints, are dominant over flexible joints.
( ) **166.** Within a white race flattened hair is nearly recessive to circular hair.
( ) **167.** The slope of the eyes of Mongolian races is due to the Mongolian fold.
( ) **168.** The greater stature of civilized white people now as compared with a generation ago is due to selective advantage of tallness

Chapter XXIV

( ) **169.** If cancer occurs in some families, not others, it should be regarded as hereditary, even if irritation is required to bring it on.

( ) **170.** Cancer occurs in some families one hundred times too often not to be inherited.
( ) **171.** The fact that goiter is influenced by the iodine content of food shows that goiter is not inherited.
( ) **172.** An infectious disease may be inherited.
( ) **173.** Mongolian deficiency is generally regarded as inherited.
( ) **174.** Huntington's chorea is dominant over normal.

Chapter XXV

( ) **175.** Inherited feeble-mindedness is strictly recessive.
( ) **176.** Epilepsy probably has in part the same genetic basis as feeble-mindedness.
( ) **177.** It is possible to recognize hereditary criminals by characteristics of the ears, nose, and facial hair.
( ) **178.** Correlation between mental ratings of parents and their children indicates that mental ability is inherited.
( ) **179.** The Tribe of Ishmael possess their characters in common because they all belong to one large relationship.
( ) **180.** Most mental qualities above the average have been accurately measured by objective methods.
( ) **181.** Feeble-mindedness, when inherited at all, is always determined by the same gene.
( ) **182.** The similarity of twins in many characters may in some instances be due to homozygosity of the parents rather than to derivation from the same egg.

Chapter XXVI

( ) **183.** A brachydactylous child could have a normal mother and normal father.
( ) **184.** If a child exhibits a typical dominant character and its mother is recessive, its father must have had the dominant character.
( ) **185.** A normal child could have a normal mother and symphalangic father.
( ) **186.** In the raising of hybrid corn for increased yield the detasseled plants furnish the seed for the main crop.
( ) **187.** Breeders' associations stipulate the economically most valuable qualities as the standards of all breeds.
( ) **188.** Any two strains of corn on crossing show about the same amount of hybrid vigor as any other two.
( ) **189.** The father of a child of blood group A, whose mother is of group AB, may be of blood group AB.
( ) **190.** A hospital which delivers a child of blood group B to two parents who are both of group O may be successfully sued for negligence.
( ) **191.** If a brachydactylous child has a normal mother and a brachydactylous man is alleged to be its father, the defendant may be adjudged guilty without further testimony.
( ) **192.** Two parents, of blood group O and group A respectively, receive from the maternity hospital a child of group B. If they sue the

hospital for alleged exchange of babies, the court should decide in their favor.

( ) **193.** It is easier to find the unknown father of a child having a rare dominant character than of a child with a common recessive character.

( ) **194.** A new plant variety reproducing only by seed has recently been patented.

Chapter XXVII

( ) **195.** It is possible to eliminate a recessive gene from a population by preventing the individuals which exhibit its character from reproducing.

( ) **196.** Eliminating the unfit in a human population should increase the number of geniuses produced.

( ) **197.** Exceptionally talented people could be produced at will if the right people were to mate as directed by experts in heredity.

( ) **198.** Feeble-mindedness can never be completely eradicated by preventing feeble-minded people from reproducing.

( ) **199.** Legislation is the principal immediate need of the eugenic movement.

( ) **200.** The aim of eugenics is to produce geniuses.

Chapter XXVIII

( ) **201.** The general birth rate is lower now than it was a generation ago.

( ) **202.** Pearl calculates that the population of the earth will not reach the saturation point for about 7000 years.

( ) **203.** It is desirable that the birth rate as a whole should be increased for the world as a whole.

( ) **204.** A population may be increasing in numbers but still not be maintaining itself.

( ) **205.** The most crowded countries of the world are the ones which are pillaging their neighbors.

Chapter XXIX

( ) **206.** Shape of head is regarded by anthropologists as a racial character.

( ) **207.** A white person 6 feet tall, with cephalic index 74, wavy light-colored hair and a fair skin is plainly a race hybrid.

( ) **208.** Blood groups are better means of distinguishing races than anatomical characters are.

( ) **209.** Anthropologists should take one or two characters to distinguish races of men, and ignore others to save confusion.

( ) **210.** A European white with tall stature, brown eyes, broad skull, and fair complexion is likely to be racially pure.

( ) **211.** American Indians belong mostly to blood group O.

( ) **212.** Negroes are increasing in numbers in the United States distinctly more rapidly than the whites.

( ) **213.** Native whites born of immigrant parents in the United States have distinctly larger families than do native whites of native parentage.

Chapter XXX

( ) **214.** There is a larger proportion of foreign-born in American custodial institutions than in the population of the United States at large.

( ) **215.** Foreign-born residents furnish more than their proportionate share of feeble-minded inmates of American custodial institutions.

( ) **216.** The present immigration law of the United States favors northwestern Europe as against southern and eastern Europe.

( ) **217.** Allotment of immigration by national quotas is probably the best possible means of selection.

( ) **218.** Admission of immigrants from any country on the basis of satisfactory family histories would benefit the quality of the population here more than does admission on the basis of nationality.

Appendix

( ) **219.** The probable error is a measure of the mistakes one makes in collecting the original facts.

( ) **220.** A mean with a small standard error gives more reliable information than one with a large standard error.

( ) **221.** The mean stature of a human population is a more reliable indication of individual statures if the population ranges from 51 to 76 inches than if the range is from 60 to 66 inches.

( ) **222.** Positive correlation between parents and offspring with respect to any variable character indicates that the character is inherited.

( ) **223.** If the coefficient of variation of an F_1 generation with respect to a quantitative character is 12.4 and that of the F_2 generation is 21.6, the character is presumably dependent on multiple genes.

PART II

Fill the blanks in the following statements with correct and significant words or expressions. In order to avoid suggesting the correct answer, some statements may be made to contain too many spaces. The extra blanks are to be left vacant; if they are filled, they are counted as errors. To ascertain the weight to be assigned to each word in an examination, divide the total weight of this part of the test by the number of blanks. The quotient is the amount to be deducted from 100 for each error or omission. Thus, if this part of a test is weighted 30, and there are 50 blank spaces, 0.6 should be deducted for either an error or no answer.

Chapter I

224. The first great student of human heredity was ________.

225. Mendel's law was rediscovered in the year ____ by ________, ____________, and ____________, all of whom found Mendel's old paper.

226. Credit for being the first scientific investigator of hybridization is often given to ______________.

227. The essential processes in Mendelism are ______________ and ______________.

228. The only biologist known to have been acquainted with Mendel's work during its progress was __________.

229. The leader of the advance in genetics in the present century has been ______________.

Chapter II

230. The most important class of organic compounds, in relation to heredity, is the group of ______________.

231. The body of protoplasm outside the nucleus of a cell is called the ______________.

232. The color of leaves in plants is due to cytoplasmic bodies called ______________, which have some relation to heredity.

Chapter III

233. Two chromosomes which contain similar genes are said to be ______________ with one another.

234. The passage of both chromosomes of a duplicated pair to one daughter cell is called ______________.

235. The minute nodules along the length of chromosomes are called ______________.

Chapter IV

236. The commonest type of reproduction in higher animals is __________ reproduction.

237. Development of an egg without entrance of a spermatozoon is called ______________.

238. Equal division of a parent in asexual reproduction is called __________, unequal division ______________.

239. To be an egg a cell must have undergone the process of ______________.

240. The two male cells of pollen fertilize, respectively, the ______ and the ______________.

Chapter V

241. The type of cleavage in a developing egg is determined by the ______________.

242. Substances which control development of neighboring parts of an embryo are known as ______________.

243. What other branch of biology has most nearly the same general aim as genetics? ______________.

Chapter VI

244. The germ cells of a male animal before maturation commences are called ______________; after growth starts but before the first division they are

_______________; after the first division they are _______________; after the second division _______________.

245. If a primary oöcyte in a given animal contains 24 chromosomes, an oögonium should contain ____, a secondary oöcyte ____, a brain cell ____, a mature egg ____, a first polar body ____, a second polar body ____. (See note on page 379.)

246. If a mature spermatozoon of a given animal contains 16 chromosomes, its spermatids contain ____, its spermatogonia ____, its skin cells ____, its primary spermatocytes ____, its secondary spermatocytes ____.

247. If an animal has 32 chromosomes, ____ of these are maternal.

248. There are ____ (number) pairs of chromosomes in the spermatids of a grasshopper.

249. One primary spermatocyte gives rise to ____ (number) spermatozoa.

250. A chromosome which is paternal in a mature egg is ________ in the individual developing from that egg.

251. When a cell with 22 chromosomes divides to produce two cells each having 11 chromosomes, that division is called ____________ division; when a cell with 14 chromosomes divides to form two cells each with 14 chromosomes, that division is ____________.

252. From 27 primary oöcytes, ____ (a number) mature eggs should be produced.

253. From 27 secondary spermatocytes, ____ spermatozoa should be produced.

254. For every 34 first polar bodies produced by a female animal there should be produced, in most species, ____ eggs.

255. In a mature egg containing 15 chromosomes, the maximum number that may be maternal is ____.

256. If the secondary oöcytes of a given species contain 9 chromosomes the primary oöcytes contain ____, the first polar bodies ____, and the second polar bodies ____.

257. Reduction division of the chromosomes in mosses takes place in the __________ generation, in the production of the __________.

258. Estimates of the number of genes in a somatic cell of Drosophila run from ____ to ____.

259. In what cells is there the greatest likelihood that genes have been seen? _______________.

260. Cells resulting from a reduction division possess the ________ number of chromosomes; an unreduced cell has the ________ number.

261. If a cell containing 18 chromosomes experiences nondisjunction of one duplicated chromosome at somatic division, the resulting daughter cells will contain ____ and ____ (numbers) chromosomes, respectively.

262. If the leaf cells of a flowering plant contain 22 chromosomes, the endosperm nuclei contain ____.

263. If the nonreproductive pollen-tube nucleus of a flowering plant contains 18 chromosomes, the generative or fertilizing nuclei of the same pollen contain ____ chromosomes.

264. If a moss spore contains 12 chromosomes, the cells of the sporophyte contain ____, those of the gametophyte ____, and the eggs ____.

Chapter VII

265. An individual whose genotype is *Aa*, or *Bb*, or *Mm* is called a ____________ ; one whose genotype is *BB* or *bb* is a ____________.

266. If a self-fertilized pink snapdragon produces 26 pink offspring, it should produce ____ ivory ones.

267. A cell formed by the union of two cells in reproduction is called a ________.

268. What mating will yield only roan cattle? ____ × ____ .

269. An animal whose genotype is *Aa* produces 600 eggs. How many of these eggs should have the formula *a*? ____.

270. If from a large number of matings of roan × roan cattle 28 white offspring are obtained, there should be ____ red ones and ____ roans from the same crosses.

271. The 36 offspring produced by *Cc* × *Cc* are ____ (a number) *CC*, ____ *Cc*, and ____ *cc*.

Chapter VIII

272. If a plant has the genotype *Bb* and it is self-fertilized, ____ per cent of its offspring should have the formula *BB*, ____ per cent the formula *Bb*.

273. An animal whose genotype is *Cc* produces 200 germ cells; ____ (a number, not a per cent) of these cells should have the formula *C*, ____ (a number) *Cc*, and ____ *c*.

274. What fraction of the phenotypically dominant plants in an F_2 generation involving just one pair of characters will breed true if self-fertilized? ____.

275. If red eye mutates to pink, and pink is found to be recessive to red, it is conventional to symbolize the red gene by the letter ____, the pink gene by the letter ____.

276. Two rose-combed fowls, A and B, bred together, produce only rose-combed offspring. Fowl B, mated with C which is also rose-combed, produces some rose-combed and some single-combed offspring. Assuming rose to be dominant over single and using *R* and *r* to symbolize the genes, the genotype of fowl A is ____, that of B is ____, that of C is ____.

277. A rat whose genotype is *Aa* produces 120 germ cells, of which __ (a number) are of formula ____, and ____ are of formula ____.

278. Two black mice bred together produce some black and some chocolate offspring. Using *Cc* as the symbols, what were the genotypes of the parents? ____ and ____.

279. What symbols would you choose to represent the genes for brown and red hair in man? ________.

Chapter IX

280. A cross between a heterozygous rough guinea pig and a smooth one yields ____ per cent ________ animals and ____ per cent ________ ones.

281. If *Cc* is mated to *CC*, ____ (a fraction) of their offspring should have the genotype ____, and ____ the genotype ____.

282. A family consisting of 65 red and 59 white offspring probably came from a mating of W __ × ____.

283. Some heterozygous animals are mated with the corresponding recessives and produce 80 offspring. Of these ____ should show the dominant character, ____ the recessive.

284. When an animal whose parents were AA and aa is mated with aa, the mating is called a __________.

285. What would you mate a black mouse with to ascertain whether it is heterozygous for brown? _______. What kind of offspring would show it to be homozygous? _______.

286. A rough-coated guinea pig mated with a smooth one yielded 4 rough and 3 smooth offspring. Using Rr for the symbols, what were the genotypes of the parents? ____ × ____.

287. The mating of an individual with a dominant phenotype to a recessive for the purpose of determining the genotype of the former is called a _______.

Chapter X

288. If green is a recessive sex-linked color in a bug, a green female crossed with a red male should produce __________ and __________.

289. If in many human families a character occurred only in males, but not in all males of the species, it would be assumed to be inherited through the __________.

290. If a male fly produces 3600 spermatozoa each containing an X chromosome, it produces ____ (a number) which have no X.

291. A homozygous red-eyed Drosophila female mated with a white-eyed male produces _____-eyed daughters and _____-eyed sons. (These colors are sex-linked and red is dominant.)

292. A white-eyed female Drosophila mated with a red-eyed male produces _____-eyed daughters and _____-eyed sons.

293. A color-blind woman who marries a normal man should have __________ daughters and __________ sons.

294. A buff-eyed female Drosophila mated with a red-eyed male yields red female and buff male offspring. If one of these F_1 red females be mated to a red-eyed male, they will produce offspring of the following eye colors and sexes: ____ _______, ____ _______, ____ _______, ____ _______.

295. A male Drosophila exhibiting a recessive sex-linked character is mated with a dominant female, and their F_1 offspring are mated together to produce an F_2 progeny of 1000 individuals. How many of these should be dominant females? ____. How many dominant males? ____.

296. The gene for a character that is handed on only from father to son for many generations is probably in the __________.

297. If a woman, herself color-blind, has 6 sons, how many of them should be color-blind? ____.

298. Black is a sex-linked character recessive to barred pattern in fowls. If a barred hen is mated with a black cock, the female F_1 offspring should be _______, the male F_1 _______.

299. If the male of a species of fly has 25 chromosomes in each body cell, the female probably has ______; if the male has 20, the female has ______.

300. If green hair is a recessive sex-linked character in a species that is usually purple-haired and has X and Y chromosomes in the male, mating a green female with a purple male should yield ________ ________ and ________ ________ offspring.

301. If bobbed (shortened bristles) occurs in homozygous but not heterozygous Drosophila females, and never in males, but the gene is transmitted to their daughters by all males whose mothers were bobbed, where is the gene which suppresses bobbed in males? ____________.

302. If some usually sex-linked recessive character in man is, as an exception in some family, transmitted from mother to daughter, the eggs of the mother must, with respect to the heterosomes, be ____ and ____

Chapter XI

303. Clumping of red corpuscles when bloods are mixed is due to ______ ______ in the red cells and ____________ in the plasma.

304. Agglutination of red cells occurs if in mixing bloods agglutinogen A is brought into contact with ____________ or agglutinogen B with ____________.

305. Blood group O mated with group AB should yield groups ____, ____, ____ among the offspring.

306. The genes for white, coral, cherry, and apricot eye color are all in the same locus of chromosome 1 in Drosophila. How many of them may exist in one normal fly? ____ Such genes are known as ________ ________.

307. If in a certain species there are four alleles (B, b, b^n, b^s) at a given locus in homologous chromosomes, the genotypes of individuals of the species with respect to the locus may be ____, ____, ____, ____, ____, ____, ____, ____, ____, ____.

308. If a gene for brown hair mutated to green, and later a gene at the same locus mutated to purple, the symbol of the last mutant gene should be ____.

Chapter XII

309. A heterozygous animal exhibiting a character which is lethal in the homozygous state, mated to another like itself, produces ____ (a number) kinds of living offspring in the ratio ________.

310. Davenport explains the absence of hemophilic women by supposing the character to be ____________.

311. If $Hh \times Hh$ yield only Hh and hh offspring, H is ________ in a ____________.

312. Individuals lacking one chromosome of a certain pair have arisen because ____________ occurred in the maturation of the egg or spermatozoon from which they developed.

313. Curly wing in Drosophila kills homozygotes in an early larval stage, homozygous creeper fowls die about ______________, homozygous

"bulldog" calves ______________, white seedling plants ________ ______________.

314. Homozygous deficiency for a segment of the X chromosome of Drosophila responsible for Bar eye is not lethal because Bar eye is due to a __________.

Chapter XIII

315. Corn that is heterozygous for starchy and sugary grain and for white and purple endosperm appears starchy and purple. If a plant thus doubly heterozygous is self-fertilized and produces an ear with 320 grains, ____ (a number) grains should be white and starchy, ____ white and sugary, ____ purple and starchy, ____ purple and sugary.

316. If *AaBb* mates with *AaBb* and they produce 96 offspring, it is expected that ____ (a number) of them will appear aB, ____ will appear ab, and ____ will appear AB.

317. Rose comb is dominant over single comb in fowls. Blue Andalusian is heterozygous for black and splashed white. A number of heterozygous rose-combed blue Andalusian fowls are mated together, and produce 160 offspring. Of these ____ should be single-combed Andalusians, ____ should be rose-combed Andalusians, ____ rose-combed black, ____ rose-combed white, ____ ________ __________, and ____ ________ __________.

318. The offspring of *PpRr* × *pprr* should be ____ per cent ______, ____ per cent ______, ____ per cent ______, ____ per cent ______, ____ per cent ______.

319. If *AAbb* is mated with *aaBB*, ____ per cent of their offspring should appear ____.

320. A tall pea plant with inflated pods, crossed with a dwarf plant having inflated pods, produces 36 tall inflated offspring, 39 dwarf inflated, 14 dwarf with constricted pods, and 12 tall constricted. The 14 dwarf constricted ones all breed true in the next generation. The genotypes of the original parents, using *Dd* and *Cc* as symbols, were ____ and ____.

321. Mating *CcDd* × *CcDd* yields ____ (a number) kinds of offspring in the proportion __:__:__:__:__:__:__:__:__, provided neither *C* nor *D* is dominant over its allele.

322. Mating *GgHh* × *GgHh* yields ____ (a number) kinds of offspring in the proportion __:__:__:__:__:__, provided *G* is dominant over its allele but *H* is neither dominant nor recessive.

323. *XxYyZz* × *XxYyZz* yields ____ (a number) kinds of visibly different offspring, derived from ____ combinations of egg and sperm, ____ (a fraction) of which combinations appear XYZ, ______ appear XyZ, and ______ appear xyz. (Assume dominance in each pair.)

324. *RrSsTt* × *rrsstt* yields ____ kinds of visibly different offspring, derived from ____ combinations of egg and sperm, ____ (a fraction) of which combinations appear RSt, ____ appear rst, ____ appear rSt.

325. If *AaBbCc* produces 200 eggs, ____ (a number) of them should be *ABC*, ____ should be *aBc*, and ____ should be *aBB*.

326. An animal which is heterozygous for 4 pairs of genes and homozygous for 2 produces ____ kinds of germ cells.

327. Self-fertilizing *MmNn* yields offspring whose genotypes are ____, ____, ____, ____, ____, ____, ____, ____, ____, ____, ____. (Give all possible genotypes, but do not repeat.)

328. The offspring of a red-eyed straight-winged Drosophila and a brown-eyed curved-winged fly are red straight. If an F_1 fly is mated with a brown curved fly and they produce 100 offspring, ____ (a number) of these should be brown straight.

329. *AaBbCcDdEe* produces how many different kinds of germ cells? ____.

330. A gray long-winged Drosophila is mated with a sooty vestigial-winged fly (*ssvv*). They produce 29 gray vestigial offspring, 32 gray long, 28 sooty long and 30 sooty vestigial. What was the genotype of the gray long-winged parent? ____.

331. If a doubly heterozygous red-eyed gray-bodied fly (*SsEe*) is mated to a doubly recessive safranin-eyed ebony-bodied fly (*ssee*) and they produce 240 offspring, ____ (a number) of these should be safranin gray and have the genotype ____.

332. A plant whose genotype is *XxYy* is self-fertilized and produces 5 offspring having the phenotype xy. How many should have the phenotype Xy? ____. How many XY? ____. How many xY? ____.

333. One parent in a cross has the genotype *ffmm*; the offspring are 26 of one kind, 21 of another, 28 of a third, 25 of a fourth. The other parent's genotype was ____.

334. In poultry, feathered shank (*F*) is dominant over clean shank (*f*); *R* produces rose, *P* produces pea comb, absence of both *R* and *P* produces single. If *ffRrPp* is crossed with *FfRrpp*, what proportion of their offspring should have both clean shanks and single combs? ____

335. If *AaBbCc* is self-fertilized and produces 128 offspring, ____ (a number) should be *AABBCC* and ____ should be *aabbcc*.

Chapter XIV

336. A fowl whose genotype is *RrPp* (the dominant genes being those for rose and pea comb) mated with another like itself produces offspring of the following kinds: ____-combed fowls whose genotypes may be ____, ____, ____ or ____; ____-combed fowls whose genotypes may be ____ or ____; ____-combed fowls whose genotypes may be ____ or ____; and ____-combed fowls whose genotype is ____.

337. If a walnut-combed fowl, mated with a single, produces only walnut offspring, its genotype is ____.

338. If a colored sweet pea *CcRr* is self-fertilized and produces 80 offspring, ____ of them should be colored, ____ white, ____ spotted.

339. If skin color in Negroes is due to 5 pairs of genes, all equally potent and all without any dominance over their alleles and with cumulative effects (like red color in wheat), there are ____ grades of mulatto with respect to color.

340. If skin color in the Negro is due to 5 pairs of genes with the properties described in the preceding problem, two parents who are hybrids between

Negro and white should produce 1 fully black child in every ____ and 1 totally white in every ____.

341. Color in sweet peas is due to a ____________ and an ____________.

342. Blending inheritance is recognized by ____________ F_1 and ______ ______ F_2. It differs from lack of dominance in the nature of the ____ generation.

343. In an F_2 generation from homozygous rose- × pea-combed fowls, there were 15 pea-combed fowls. How many walnuts should there be? ______. How many single? ______.

344. Suffolk hornless female sheep mated with Dorset horned males produce ________ ________ and ________ ________ offspring.

345. If wheat color is due to three pairs of genes as Nilsson-Ehle describes, there are ______ (a number) red or reddish plants out of every ______ in an F_2 generation derived from a cross red × white. If the genes are all equal, cumulative, and not dominant or recessive, the red and reddish wheats should fall into ______ (a number) visibly different classes.

346. If a red flower crossed with a white one yields pink offspring, and if these pink ones on being self-fertilized yield almost all pink offspring ranging from nearly white to nearly red, with an occasional white and red, the inheritance of flower color is ____________.

347. A yellow squash (*wwYY*) is crossed with a white one (*Wwyy*). Of their 180 offspring ____ (number) should be white and of the genotype

348. In corn, *C* and *R* are necessary for color of endosperm, which is red in the absence of any modifying gene. If *P* is likewise present with *C* and *R*, the endosperm is purple. With either *cc* or *rr* the endosperm is white. If red corn *CcRrpp* is crossed with white *ccRrPp*, among their 80 offspring ______ (number) are white.

349. If baldness is dominant in men and recessive in women, as some have held, and a bald brown-eyed man (whose father was non-bald and blue-eyed) marries a non-bald blue-eyed woman (whose father and numerous brothers were all bald), their first child has ____ chances in ____ of becoming a bald blue-eyed man.

350. A white female clover butterfly *Ww* mated with a yellow male *Ww* produces 56 offspring of which ____ (number) should be yellow females, ____ yellow males, ____ white females, ____ white males.

Chapter XV

351. If an organism possesses two pairs of genes, the dominant of either of which alone produces a certain character, and both together produce the same character, these genes are called ________________.

352. If a true-breeding shepherd's-purse with triangular capsules is pollinated by one with spindle-shaped capsules, the F_1 has ____________ capsules; and if these F_1 plants, self-fertilized, produce 64 F_2 offspring, ____ of these should have triangular capsules, ____ spindle-shaped.

353. If plants *AAbb*, *aaBB*, *AABB*, and *AaBb* are all indistinguishable from one another, but are all different from *aabb*, *A* and *B* are ________

354. If a triangular-capsuled shepherd's-purse of genotype *Ccdd* is pollinated by a triangular *CcDd* and they produce 80 offspring, ___ (number) should have spindle-shaped capsules, ___ triangular.

Chapter XVI

355. Primulas of a red-flowered race have ___ flowers at 20°C., ___ flowers at 35°C. Their seeds produced at 35°C. yield ___-flowered plants if reared at 20° and ___-flowered plants if reared at 35°C.

356. A barley variety may be ___ if reared at low temperature, ___ at high temperature.

357. Abnormal abdomen in Drosophila is dependent on ___ and ___.

358. A gland whose secretion must diffuse out instead of flowing out through a channel is known as a ductless or ___ gland

359. Wing development in aphids may be suppressed by ___.

Chapter XVII

360. Six pairs of chromosomes may be arranged in ___ ways on the reduction spindle, and ___ (a number) kinds of germ cells, each with a different combination of chromosomes, can be produced as a result.

361. If the chance that A shall happen is 1 in 2, and the chance that B shall happen is 1 in 3, the chance that A and B shall happen together is 1 in ___.

362. A brown-eyed man marries a blue-eyed woman, and their first child is blue-eyed. What is the chance that the second child will be blue-eyed? ___ in ___.

363. If 60 dice are thrown, ___ (a number) would be expected to turn up the 6-spot, ___ the 4-spot.

364. If a pyramid has four equal triangular faces numbered 1, 2, 3, 4, and two such pyramids are thrown simultaneously, how many throws would you expect to make in order to have both pyramids rest on the 3-face simultaneously one time? ___.

365. Five pairs of chromosomes may be arranged in ___ (a number) different ways on the spindle of the reduction division.

366. In Fig. 90, what is the chance that the man numbered 3 is a homozygous normal? ___ in ___.

367. In Fig. 91, if III-3 and III-6 marry, what is the chance that any given child of theirs will be not only normal but free from the undesirable recessive gene?. ___ in ___.

Chapter XVIII

368. If an animal has the genotype *ABC-abc*, and the genes are arranged in the chromosome in the order in which they are here named, one of its germ cells having the formula *AbC* results from a ___ crossing over.

369. A number of spotted short-haired rabbits whose mothers were self-colored and long-haired were mated to self-colored long-haired rabbits, and

produced 26 spotted short-haired, 4 spotted long-haired, ____ self-colored short-haired, and ____ self-colored long-haired offspring.

370. Mating *RS-rs* × *rrss* yielded 100 offspring, of which 43 had the genotype ______, ____ the genotype ______, ____ the genotype ______, and ____ the genotype ______.

371. If *CcDd* × *ccdd* produce 100 offspring, of which 14 are cD, the genotype of the doubly heterozygous parent should be written ______ to show the linkage relation of the two pairs of genes.

372. If *MmPp* × *mmpp* produce 160 offspring of four classes, numbering 39, 42, 38, and 41, respectively, how many pairs of chromosomes are required to contain the genes named? ____.

373. A male Drosophila, *Tw-tW*, mated with *ttww*, produces 86 offspring appearing ____, ____ appearing ____, ____ appearing ____, and ____ appearing ____.

374. If an animal whose genotype is $(Ab)(Ab)$ is mated with $(aB)(aB)$, which of the characters will show in F_1, using only one letter of each pair to indicate the visible character? ____. What will be the genotype of F_1, using parentheses to indicate the genes in each chromosome? ______. How many kinds of germ cells will the F_1 produce? ____. If crossing over occurs in 30 per cent of all cells and a total of 400 germ cells is produced, how many of these germ cells will have the formula *Ab*? ______. How many *ab*? ______. How many *aB*? ______. How many *AB*? ______.

375. If $(XY)(xy)$, in which 20 per cent of crossing over occurs, is mated to $(xy)(xy)$ and 600 offspring are produced, how many of these offspring should be $(XY)(xy)$? ____. How many $(Xy)(xy)$? ____. $(Xy)(xY)$? ____.

376. An animal which is homozygous for *CD* is mated to one homozygous for *cd*, and the F_1 is backcrossed to the double recessive. Of the backcross generation 604 have the appearance CD; 596 have the appearance cd; 97 Cd; 103 cD. In what percentage of the primary oöcytes and spermatocytes do the chromosomes break between *C* and *D* (or between *c* and *d*)? ____.

377. A homozygous spotted short-haired rabbit mated to a homozygous self-colored Angora rabbit produced spotted short-haired offspring. The F_1 animals were crossed back to self-colored Angora rabbits and produced 16 self-colored short haired, 14 spotted Angora, 144 self-colored Angora, and 139 spotted short haired. What percentage of crossing over occurs between the two pairs of genes? ______.

378. If *CcDd* × *ccdd* yield offspring in the following numbers: 28, 32, 94, 99; and if *FfGg* × *ffgg* yield 62, 66, 12, 10; the genes *Cc* and *Dd* are ____ times as far apart as are the genes *Ff* and *Gg*. (An answer to the nearest one decimal place is sufficient.)

379. A male Drosophila whose genotype is $(MN)(mn)$ produces germ cells whose formulas are ____, ____, ____, ____. A male rabbit having the same genotype produces spermatozoa having the formulas ____, ____, ____, ____.

380. (*a*) A certain tall spherical-fruited tomato plant which is heterozygous for dwarf stem and pear-shaped fruit crossed with a dwarf pear-fruited plant produces 81 tall spherical, 79 dwarf pear, 22 tall pear, and 17 dwarf spherical. (*b*) Another tall spherical plant crossed with a dwarf pear produces 21 tall pear, 18 dwarf spherical, 5 tall spherical, 4 dwarf pear.

Using parentheses to indicate linkage, what is the genotype of the first plant in (*a*)? ______. The first plant in (*b*)? ______.

381. If $(Ab)(aB)$ crossed with $(ab)(ab)$ yields 68 offspring appearing Ab, 64 aB, 33AB, and 35 ab; and if $(AC)(ac) \times (ac)(ac)$ yield 79 AC, 87 ac, 15 Ac, 19aC; and if A is 20 arbitrary units of measure distant from C; then A is ____ (a number) of the same units distant from b.

382. If crossing over between R and S is 15 per cent, that between S and T is 22 per cent, and that between R and T is 7 per cent, indicate the order of the genes and the distances between them in the chromosomes. ____________.

383. If a mammal Xy-xY is crossed with Xy-xY and there is 20 per cent of crossing over, the offspring will consist of ____ per cent phenotypically XY, ____ per cent phenotypically xY, ____ per cent Xy, and ____ per cent xy.

Chapter XIX

384. That part of the cycle of a moss which has two genes for each character is called the ________, while the part that has only one gene is the

385. What organism has more linkage groups of inherited characters than it has pairs of chromosomes? ______.

386. Sex-linked characters are transmitted from generation to generation in the same manner as ____________.

387. A chromosome aberration which results in gene A being linked with genes of which it was previously independent is called a ____________.

388. A chromosome aberration which changes the distance between gene A and other genes with which it has been, and still is, linked is either ____________ or ____________.

389. The location of genes in the bands of the salivary gland chromosomes of Drosophila has been discovered by means of overlapping ____________.

Chapter XX

390. Heredity is called ____________ if its units behave in the same way as chromosomes behave.

391. A snail whose genotype is Ll with respect to direction of coiling produces eggs whose formula is ____ which grow up into ________-coiled snails, and eggs whose formula is ____ which develop into ________-coiled snails.

392. A female flower on a variegated branch of a four-o'clock is pollinated from a flower on a green branch of the same plant. The seeds from this cross yield ________ offspring.

393. Whether a snail shell coils left or right depends on the genes in the snail's ________.

394. What character in corn was judged cytoplasmic because removal of the various chromosomes by hybridization did not remove it? ________.

395. ________ are known, and ____________ have been suggested, to be the mechanism of non-Mendelian heredity.

Chapter XXI

396. A honeybee developed from a fertilized egg is a ________; one developed from an unfertilized egg is a ________.

397. A female honeybee has ____ (a number) chromosomes in her body cells, a male has ____.

398. The mature eggs of a honeybee contain ____ (a number) chromosomes, the mature spermatozoa ____.

399. Two individuals coming from one egg in man are called ______________ ______.

400. Secondary sexual characters in mammals are due to ____________.

401. A young Bonellia becomes a male by being ________________.

402. Individuals showing an irregular mixture of typical male and female parts are called ____________.

403. There are ____ (a number) chromosomes in the body cells of a man, ____ in the body cells of a woman.

404. The normal change of sex in ____________ is governed by chemical substances diffusing out of older individuals.

405. In plants ____ (number) species are known to have their sex related to specific chromosomes.

406. Nondisjunction of the ____ chromosome in embryonic cleavage might explain gynandromorphs.

407. Insect broods of multiple embryos which include both sexes are probably due to ____________.

408. Individuals intermediate between males and females are called ____________.

Chapter XXII

409. Mutations have been artificially produced by ______, ________, and ______ (agents).

410. Mutations arose in Muller's X-rayed flies ____ times as often as in the controls.

411. To believe that children of older parents are genetically superior to those of younger parents requires a belief in __________________________.

412. Fox found the lengthening of siphons in Ciona to be due to ______ ______; Kammerer had thought it was due to ____________.

413. What organism extensively bred under close observation has produced no mutation? ______.

414. What chromosome change may most quickly yield a new species? ____________________.

415. If in one generation 20 per cent of chromosomes 1 of a species contain gene *a* at a given locus, and 80 per cent contain *A*, the expectation is that the next generation will have ____ per cent *a* and ____ per cent *A* at that locus.

416. The nature of the gene which arises by mutation from gene *B* is determined by ________________.

417. If a given gene locus in a species is occupied 70 per cent by gene *H* and 30 per cent by *h*, ____ per cent of the individuals of the species should have the genotype *hh*, ____ per cent *Hh*, and ____ per cent *HH*.

418. Reducing the percentage of gene M at a given locus in the chromosomes of a species from 90 per cent to 80 per cent reduces the frequency of MM individuals from ____ per cent to ____ per cent.

419. The only advantage in evolution which a character can confer on its possessor is to enable it to ____________________.

Chapter XXIII

420. Failure of the halves of the jaw to meet in fetal development causes ____________ and ____________.

421. If blue eye is strictly recessive to brown, and two blue-eyed parents have a brown-eyed child, one of the parents may have had a gene for ________ plus an ____________ gene.

422. Suppose that brown eye in man is strictly dominant and that no inhibitor is present in a family in which the father is heterozygous, the mother blue-eyed. If their first three children are brown-eyed, what is the chance that the fourth will be blue? ________.

423. If there are six alleles at the locus of the basic hair color gene in man (Lenz's scheme, page 250) and these have a dominance order in which each gene is dominant over all those below it in the series, how many grades of hair color may there be with respect to this locus alone? ______.

424. If a woman with free ear lobes, whose mother had adherent lobes, marries a man with free ear lobes, whose father had adherent lobes, what is the chance that their first child will have adherent lobes? _____.

Chapter XXIV

425. Excessive sensitiveness to certain substances is called ________.

426. A migrainous woman whose relatives all have normal vision and whose mother is free from migraine marries a day-blind man whose relatives are free from allergy. What is the expected nature of their children? ________________.

Chapter XXV

427. Notorious families of criminals and mental defectives are the ______, ________, ________, ________, ________.

428. The commonest form of insanity is called ____________; its mode of inheritance is ________________.

429. Hurst's scheme of inheritance of intelligence involves ____ (number) pairs of genes, ____ (number) of which are modifiers.

430. Musical ability is made up of ________, ________, ________, ________, ________.

Chapter XXV

431. In synthesizing a new variety of tobacco, suppose two desired recessive characters are obtainable in one variety, and a third (dominant) character is available in another. If these varieties are crossed, and the breeding is carried to an F_2 generation of 384 plants, how many of these plants should have the required combination of characters? ____. How many of the desired type should breed true on self-fertilization? ____.

432. Vigor in corn is increased by ____________.

Chapter XXVII

433. Preventing all feeble-minded persons from reproducing should reduce the number of feeble-minded by ____ per cent in one generation, by ____ per cent in two generations, by ____ per cent in three generations, and by ____ per cent in 22 generations.

434. The greatest proportion of sterilization operations in the United States have been performed in the state of ____________.

435. The only country which has performed more sterilization operations than the United States is ____________.

436. Deliberate eugenic control of marriages by authority was once in effect in ________________ (locality).

437. In the cities of ____________, ________ and ________ the birth rate in the proletarian districts is now almost as low as in the wealthy sections.

Chapter XXVIII

438. Various calculations place the date of saturation of population of the United States at the years ______, ______, ______.

439. Cities of the state of ____________ fall farther short of maintaining their population than those of any other state.

440. Of all European countries ____________ lacks the most of maintaining its population at present birth rates.

441. Pearl's estimate of the ultimate population of the world is ______ ______, to be reached by the year ______.

Chapter XXIX

442. A head 8 inches from front to back and 6 inches wide has a cephalic index of ______.

443. The Swedish people are largely of the ______ race, Spanish people of the ________ race, and Hungarians ________.

444. The pure ________ white race has high stature, hairy body, ________ complexion, ________ eyes, and ________ skull.

445. The main racial divisions of mankind are the ________, ________, and ____________.

446. The greatest proportion of white people in the United States are derived from ________________ (country), the next greatest from ________.

Chapter XXX

447. Immigration into the United States was negligible until the year ______; it reached its peak in the year ______.

448. The total immigration into the United States permitted by the present law is ________ (a number) plus ________.

449. The most common defect for which people are committed to institutions is ____________.

450. Ireland, according to Laughlin's studies, is the least satisfactory place to draw immigrants from with respect to ________, ________ the worst country with respect to crime, ________ the worst with respect to blindness, and ________ with respect to all defects combined.

Appendix

451. Usually the first thing to be ascertained concerning a quantitative character that is or may be different in two populations is the ______.

452. How much information a mean gives regarding a population is shown by the ________________.

453. If weight of men increases with increase of stature, that fact would best be shown statistically by the ________________.

454. The highest possible value of a coefficient of correlation is ____; the lowest possible value is ____.

455. How variable a population is, is shown by its ________ ____________.

456. If the mean of one population is 17.1 ± 0.03, and that of another population is 16.2 ± 0.04, the difference between the means is ______ ± ______.

457. If the mean of a random sample is 25 ± 2, there is a ______ chance that the real mean of the entire population may be as high as 27 or as low as 23.

458. The standard error of an F_2 distribution of 326:104 is ______.

459. A probable error is ______ times the corresponding standard error.

PART III

Underline each word or expression in parenthesis which will, if inserted by itself, make the statement correct and significant. In case of uncertainty, unless you wish to gamble, place a question mark directly on the word. More than one word, or even all of them, may be correct; likewise, all may be wrong. It is important to consider each word separately. Read the sentence through with one of the alternative expressions inserted; if the sentence is correct and significant, underline that expression. Then read similarly with each of the other words inserted in turn. Error is likely to result in some instances if all or several of the alternative words are kept in mind at the same time. To determine the weight to be assigned to each word or expression in an examination, divide the weight of this part of the test by the total number of words or expressions in parenthesis. The quotient is the amount which should be deducted from 100 for each question mark; twice that amount should be deducted for each word underlined when it should not be or not underlined if it should be. For example, if multiple choice questions are weighted 30 in the examination, and there are 150 expressions in parenthesis, $\frac{1}{5}$ should be deducted for each question mark and $\frac{2}{5}$ for each mistake.

Chapter I

460. The earliest records of (wheat, rice, oats, soybean, rye, cotton) show that it was then a wild plant.

461. Mendel contrasted (one whole plant, one character) with another in his crosses.

462. It would have been (easier, harder, neither easier nor harder) to discover Mendel's law in man than in peas.

463. Sex was known earlier in (animals, plants) than in (animals, plants).

464. Darwin (criticized, adopted, never heard of) Mendel's laws.

465. Ideas of evolution and natural selection (hastened, retarded, had no influence on) the acceptance of Mendel's discoveries.

466. Mendel's scientific work was (advanced, hindered) by his (administrative duties, controversy over taxation, correspondence with Nägeli).

Chapter II

467. Cells govern the (physiology, development, heredity) of (animals, plants).

468. (Hair, skin, bone) owes its characteristics to cells.

469. Cells work (singly, cooperatively) in producing hereditary characters.

Chapter III

470. The two chromosomes derived from one chromosome in cell division are (equal, unequal).

471. A chromosome divided (lengthwise, crosswise) would yield two equal chromosomes.

472. The units making up a chromosome are arranged in a (rectangle, row, circle, cube.)

473. Chromosomes are (hypothetical, actual, visible, invisible) objects.

Chapter IV

474. (All, some, no) sexual reproduction includes the production of true germ cells.

475. Parthenogenesis is (sexual, asexual) reproduction.

476. Sexual reproduction offers (more, fewer) opportunities of bringing about recombinations of characters than asexual reproduction does.

477. Horticultural varieties are more likely to retain their characters with (sexual, asexual) reproduction than with (sexual, asexual).

478. Asexual reproduction probably arose (earlier, later) in the evolution of living things than did sexual reproduction.

Chapter V

479. The different parts of an organism have their fate decided (all at the same time, at different times) in development.

480. The cells of insects have (more, less) autonomy in development than do the cells of vertebrate animals.

481. The anterior parts of embryos generally develop (earlier, later) than the posterior parts.

Chapter VI

482. If a muscle cell of a given animal contains 18 chromosomes, its primary oöcytes may contain (1, 4, 9, 12) maternal chromosomes, and its secondary oöcytes may contain (1, 4, 9, 12) maternal chromosomes. (See note on page 379.)

483. The mature germ cells of an animal (may, must, cannot) contain as many maternal chromosomes as its somatic cells contain.

484. A (primary oöcyte, mature egg, oögonium, secondary oöcyte, first polar body, second polar body) may contain just 9 chromosomes.

485. A (primary oöcyte, mature egg, oogonium, secondary oöcyte, first polar body, second polar body) may contain only (maternal, paternal) chromosomes.

486. Maturation in the female is like that in the male with respect to the (chromosomes, cell body).

487. Most animals have an (even, odd) number of chromosomes in their body cells.

488. If a bone cell contains 10 maternal chromosomes, a mature egg of the same animal may contain (3, 6, 10, 14, 18) maternal chromosomes.

489. Of 26 chromosomes in a secondary spermatocyte (9, 13, 26) may be maternal.

490. There are (twice, just, half) as many chromosomes in a first polar body as in a second polar body in the same female.

491. The maternal chromosomes in a spermatozoon are (maternal, paternal) in the individual developing from the egg which that spermatozoon fertilizes.

492. The formation of gametes follows (more, less) quickly after the reduction division in mosses than in flowering plants.

493. (More, fewer) cell divisions occur between the reduction division and the production of gametes in flowering plants than in animals.

494. The cells of a moss (gametophyte, sporophyte) must contain equal numbers of maternal and paternal chromosomes.

495. If an oöcyte contains 9 chromosomes, it is a (primary, secondary) oöcyte.

496. One chromosome (may, must, cannot) be exactly like one other chromosome in the same body cell.

Chapter VII

497. (Red, pink, ivory) snapdragons breed true if self-fertilized.

498. (Pink, red, ivory) snapdragons and (red, white, roan) cattle are heterozygotes.

499. (Homozygotes, heterozygotes) breed true if (self-fertilized, mated to others like themselves).

500. A man who is heterozygous for two blood agglutinogens possesses (one, two) agglutinogens.

Chapter VIII

501. An F_2 ratio of 3:1 indicates that their grandparents differed in (1, 2, 3 4) characters.

502. Segregation of genes occurs in (heterozygotes, homozygotes).

503. Reduction division of a pair of chromosomes results in segregation of (all, half, none) of the genes contained in them.

504. The (new, old, dominant, recessive) character of any pair suggests the symbol to represent the genes, while (age, dominance) determines whether a capital or small letter be used.

505. Mating Aa with Aa may result in a ratio of (1:1, 1:2:1, 3:1, 9:3:3:1) among the offspring.

506. If in a mixed population every time two individuals showing character X mate their offspring all show X, X is a (dominant, recessive) character.

Chapter IX

507. To obtain a ratio of 1:1 from a given mating in which the genotype of one parent is mm, the genotype of the other parent must be (MM, Mm, mm).

508. To discover whether a phenotypically dominant individual is Aa or AA, it should be mated to (AA, Aa, aa).

509. An animal whose genotype is Gg mated with gg may produce 25 offspring of one kind and (7, 23, 61) of another.

Chapter X

510. A (female, male) (bug, mammal) has two X chromosomes.

511. A mature germ cell containing a W chromosome is (a, an) (egg, sperm); one containing a Y chromosome is (a, an) (egg, sperm).

512. The genes for sex-linked characters in (flies, man) are in the (X, Y, Z, W) chromosomes.

513. A male fly inherits his sex-linked characters from his (mother, father).

514. A female mammal inherits her sex-linked characters from her (mother, father).

515. A recessive sex-linked character shows in a (male, female) fly which has only one gene for it in each cell.

516. If a female fly showing a recessive character is mated with a male showing the alternative dominant character, one can *first* tell in the (F_1, F_2) generation whether the character is sex-linked.

517. If the F_2 generation gives the first indication that a moth character is sex-linked, the original mating was between a recessive (male, female) and a dominant (male, female).

518. If a mating between a dominant female and a recessive male yields F_1 females of one kind and males of the other, the animals used may be (birds, flies, mammals, moths).

519. A gene in a W chromosome is transmitted by (one, both) parents to (some, all) of the offspring.

520. A female bug may have (18, 19, 20, 21, 22) chromosomes in each body cell.

521. A male (butterfly, bird, fly, mammal) may have (23, 25, 29) chromosomes in each body cell.

522. A male bug having 24 chromosomes in its body cells has (a, an) (X, Y) chromosome; one having 25 chromosomes in its body cells has (a, an) (X, Y) chromosome.

523. Male (flies, mammals, birds) always show any characters for which they have any, even one, sex-linked gene, even if the gene is recessive.

524. If in Drosophila a (long, miniature)-winged female mated with a (long, miniature)-winged male produces miniature males and long-winged females, miniature wing is a recessive sex-linked character.

525. A fly whose genotype for a sex-linked character is M is a (male, female).

526. A (mammal, moth) with 55 chromosomes in its body cells must be a female.

527. If the gene for xeroderma pigmentosum in man is in the X chromosome, females should (sometimes, never) have the disease; if it is in the Y chromosome, females should (sometimes, never) be afflicted.

Chapter XI

528. To make tests of the blood groups it is most useful to have on hand prepared sera of groups (O, A, B, AB).

529. A mouse whose genotype is A^Ya^t is phenotypically (yellow, black and tan, intermediate); a fly whose genotype is w^+w^a has (wild-type red, apricot, intermediate) eyes; a fly whose genotype is ww^c has (white, cherry, pale cherry) eyes.

Chapter XII

530. An organism lacking one chromosome of a pair, even a small one, is likely to (die, be structurally different); if it lacks both chromosomes of one pair, it is apt to (die, be structurally different).

531. Absence of certain genes from both chromosomes of a pair is (usually, seldom, never, always) fatal to the individual harboring the deficiency.

532. If a homozygous deficiency is not fatal, the missing genes may be (unessential, repeated elsewhere in the chromosomes).

533. The gene for (Star eye, yellow body) in Drosophila and that for (yellow coat, spotting) in mice are lethal in the homozygous condition.

534. The character Dichaete which involves spread wings and absence of certain bristles in Drosophila is (dominant or partially dominant, recessive) for the visible effect and (dominant or partially dominant, recessive) for a lethal effect.

535. (Color blindness, hemophilia, brachyphalangy) in man has been suggested to be lethal in homozygotes.

Chapter XIII

536. An animal whose genotype is $EeFfGg$ produces 240 germ cells, of which (16, 20, 30, 40, 80) have the formula (eFG, EFG); (20, 30, 40, 80) have the formula (eGG, EfG); and (20, 30, 40, 80) have the formula (EFg, eeF).

537. In an F_2 generation from a dihybrid cross 122 plants are produced, of which 8 are red-flowered and tall. Red is (dominant, recessive) and tallness is (dominant, recessive).

538. When an F_2 generation consists of two kinds of individuals in the ratio of 3:1, the original parents differed in (1, 2, 3, 4) pairs of genes.

539. $MmSs$ should produce (1, 2, 4, 8) kinds of germ cells.

540. If $AaBb$ is crossed with $AABb$, some of the offspring should be ($AABB$, $aaBb$, $Aabb$, $aabb$).

541. If $SsTt$ is mated with $sstt$, and they produce 200 offspring (21, 35, 49, 66, 97) might have the phenotype St.

542. Barred pattern is sex-linked and dominant over black in poultry, and feathered shank is autosomal and dominant over clean shank. If a black, clean hen is mated with a barred, feathered cock and their offspring are bred among themselves, all of the (barred, clean; black, clean; black, feathered; barred, feathered) members of F_2 will be (females, males).

Chapter XIV

543. If A, B, and C determine color in wheat, are cumulative and equal in effect, and exhibit no dominance, $AABbCc$ is (darker, lighter) red than ($AabbCC$), and $aaBBCC$ is (darker, lighter) than $AaBbCc$.

544. A mouse $CCaaBbddPpss$ is (spotted dilute black, pink-eyed spotted brown).

545. A female clover butterfly Ww is (white, yellow); a male WW is (white, yellow).

546. A fowl $RRpp$ is (rose, walnut, pea, single); one which is $RrPP$ is (rose, walnut, pea, single).

547. Among the progeny of fowls $rrPP \times RrPp$ are some (walnut, pea, rose, single)-combed birds.

548. Corn of the genotype $CcRRpp$ is (white, red, purple); $CcrrPP$ is (white, red, purple); $ccRRPp$ is (white, red, purple); and $CCRrPp$ is (white, red, purple).

Chapter XV

549. In the deer mouse, *Peromyscus maniculatus*, color is dependent on a dominant gene C. Lack of C makes the mouse albino. If the gene Y is present in addition to C, the mouse is the wild-type gray, but Y has no effect by itself. The gene y with C produces yellow; with c it has no effect. A mouse of genotype $Ccyy$ is (albino, yellow, gray); $ccYy$ is (albino, yellow, gray); $CCYY$ is (albino, yellow, gray); and among the 96 offspring derived from $CcYy \times CcYy$ matings (6, 18, 24, 40, 54) should be albino, and (6, 18, 24, 40, 54) should be yellow.

550. In sweet peas a white flower may have the genotype ($CcrrBB$, $CCRrbb$, $ccRRbb$, $CCrrbb$, $CCRrBB$).

551. The 27/64 class in an F_2 generation always possesses a (dominant, recessive) gene in each of (one, two, three, four) pairs.

Chapter XVI

552. If a red-flowered Chinese primula be reared at a temperature of 35°C. for a few days, its (flowers open white, genes mutate to white).

553. The twisting of teasel stems is determined by (genes, temperature, moisture, nutrition).

554. Extra legs in Drosophila are due to (genes, temperature, moisture, nutrition).

555. Abnormal abdomen in Drosophila is due to (genes, temperature, moisture, nutrition).

556. In order to produce red flowers in primulas (a gene for red color, low or moderate temperature) is necessary.

557. The Bar eye of Drosophila is made (larger, smaller) by (high temperature, abundant nutrition).

558. (More, fewer) genetic varieties of organisms have been imitated by treatment with chemical substances than by treatment with temperature.

559. (Eye color, stature, fingerprint pattern) in man is easily modifiable by environment.

560. Among 300 throws a legitimate die might turn up the 6-spot (20, 47, 88) times.

Chapter XVII

561. Among events connected with heredity which are determined by chance are (position of chromosome pair on reduction spindle, relative number of *B* and *b* spermatozoa produced by *Bb* male, number of *A* and *a* eggs produced by *Aa* female).

562. The chance that of two pairs of chromosomes the maternal member of both will turn toward the right end of the reduction spindle is 1 in (2, 4, 5, 6, 8).

563. To produce 16 kinds of germ cells an animal must be (heterozygous, homozygous) for (1, 2, 3, 4, 5, 6, 7, 8) pairs of genes.

564. Dr. Carothers (reasoned from experiments, saw in the microscope) that chromosome pairs are placed in all possible relations to one another on the reduction spindle.

565. Into the germ cells of *GgHh*, *G* is (more, less, neither more nor less) likely to go with *h* than with *H*, and *g* is (more, less, neither more nor less) likely to go with *H* than with *h*.

566. Assuming that all normally functional cells survive one would conclude that an *Aa* female may produce 64 *A* eggs and (61, 64, 66) *a* eggs.

567. Four pairs of chromosomes may be arranged in (2, 4, 8, 16) ways on the spindle of the reduction division.

Chapter XVIII

568. If *Bc-bC* × *bbcc* yield 28 offspring showing one dominant and one recessive character, they might have (9, 28, 55) offspring showing both dominants.

569. If an animal whose genotype is *AaBb* produces 40 germ cells *AB* and 60 *Ab*, it should produce (20, 40, 50, 60, 240) germ cells *aB*.

570. A male Drosophila whose formula is *GH-gh* produces spermatozoa of the following kinds: (*GH*, *gh*, *gH*, *Gh*).

571. An animal whose genotype is *AaBb* mated with *aabb* might produce offspring in the ratio (1:1:1:1, 6:1:1:6, 9:3:3:1, 1:1, 5:2:2:17).

572. If *AaBb* × *aabb* produces 68 AB-appearing offspring, 65 Ab, 67 ab, and 69 aB, the two pairs of genes are in (the same, different) pair(s) of chromosomes.

573. Mating *AaBb* × *aabb* should result in equal numbers of the various classes of offspring if the two pairs of genes are in (the same, different) pair(s) of chromosomes.

574. A (male, female) Drosophila, *Xy-xY*, mated with *xxyy*, produces 88 offspring of one kind in a total of 176.

Chapter XIX

575. The (sporophyte, gametophyte) of a moss may show a heterozygous condition.

576. A moss gametophyte has the (haploid, diploid) number of chromosomes in its cells.

577. A moss (sporophyte, gametophyte) has two genes for each character.

578. A moss gametophyte can produce (1, 2) kind(s) of spermatozoids with respect to a given character; the sporophyte can produce (1, 2) kind(s) of spores with respect to the same character.

579. An organism developing from an unfertilized egg which has undergone reduction should have (1, 2) gene(s) at each locus.

580. Cell division in which nondisjunction occurs results in a cell having one (more, fewer) chromosome than normal.

Chapter XX

581. The color of a silkworm moth's egg is determined by the genes in the (egg, moth that laid it, male moth that fertilized it).

582. To ascertain the probable genes for egg-shell color possessed by a moth, the color of the egg (from which she hatched, which she lays) should be observed.

583. A snail whose genotype is *Ll* with respect to direction of coiling (may, must) have a (dextral, sinistral) shell.

584. If *L* represents dextral shell, *l* sinistral, the genotype of a snail occupying a dextral shell may be (*LL*, *Ll*, *ll*). The genotype of its mother may have been (*LL*, *Ll*, *ll*).

585. Non-Mendelian characters may (gradually disappear, continue undiminished) through a number of generations.

Chapter XXI

586. Multiple embryos, in order to be of the same sex, must have their sex determined (at the time of fertilization, before the separate development of the several embryos begins).

587. (Combs, tail feathers, spurs, crowing) are secondary sexual characters in fowls.

588. The (germ, interstitial) cells in (ovaries, testes) are believed to produce the hormone that develops secondary sexual characters.

589. X-ray treatment injures (germ, interstitial) cells more than (germ, interstitial) cells.

590. The (shape, pattern) of certain chicken feathers is determined by genes, while the (shape, pattern) is determined by a hormone.

591. Grafting a piece of chicken skin when young to a bird of the opposite sex may change the (shape, pattern) of the feathers.

592. Chemical substances in the water usually (increase, diminish, have no effect on) the number of males in rotifers.

593. Sex may be determined partly by environment in (rotifers, man, Crepidula, cattle).

594. (Shape, barred pattern) of feathers is shown by grafting experiments to be a secondary sex character in fowls.

595. Intersexes in Drosophila indicate that femaleness is favored by the (X, 2d, 3d, 4th) chromosome and maleness by the (X, 2d, 3d, 4th).

596. Gynandromorphs may be due to (binucleate eggs, localized environmental influence, nondisjunction of a chromosome pair).

Chapter XXII

597. Evolution occurs through (recombination, mutation).

598. Evolution by recombination requires (previous mutations, sexual reproduction, asexual reproduction).

599. Natural radiation from the earth may produce (all, some) of the mutations that occur.

600. Among the reasons for partial or complete sterility of species hybrids is (dissimilarity of chromosomes, sterility genes, structural differences in the genitalia).

601. The direction which evolution takes depends on (chance, direction of mutation, migration, selection).

Chapter XXIII

602. Albinism is (recessive, dominant) to skin color in man.

603. Spotted skin is (dominant, recessive) to uniform color.

604. A person heterozygous for extra fingers (always, sometimes, never) has extra fingers.

605. Curly hair in white people is approximately (dominant, recessive) to straight.

606. A hair that is (circular, elliptical) in cross section is straight.

607. A brachydactylous father (may, must, cannot) have brachydactylous children.

608. Ptosis is a (muscle, nerve) defect, (dominant, recessive) to the normal

Chapter XXIV

609. Color-blind women are (more, less, neither more nor less) numerous than would be expected if color blindness is sex-linked.

610. (Asthma, anemia, hay fever, edema, eczema, elephantiasis) is a form of allergy.

611. Huntington's chorea is (recessive, dominant, irregularly dominant).

Chapter XXV

612. The sons of (honor, pass) men from Oxford are mentally superior to the sons of (honor, pass) men.

613. Heredity is (more, less) likely to account for differences between children of the same parents than environment is.

614. Mental tests have (often, sometimes, never) shown that identical twins are markedly different from one another.

615. If feeble-mindedness is a simple recessive character, (all, some, none) of the children of two feeble-minded parents should be normal.

616. Seashore devised a method of measuring (literary, musical, mechanical) ability.

617. A person's rating in musical tests improves with (age, musical training).

618. Underscore the one of the following psychological measures which you regard as most reliable: (intelligence quotient, Banker's student's ability index, social quotient, Seashore's musical ability score).

Chapter XXVI

619. A child with normal hands whose mother has normal hands (may, must, cannot) have a father with (normal, brachydactylous) hands.

620. A child of blood group O, whose mother is of blood group O, may have a father of blood group (O, A, B, AB).

621. The best criterion of an animal's worth as a breeder is its (own, relatives') qualities.

622. A normal-fingered child whose mother is normal (may, must, cannot) have a brachydactylous father.

623. A character, to be frequently useful in deciding cases of disputed parentage, should be (dominant, recessive) and (common, rare) in the population, and should have its mode of inheritance (exactly, approximately) understood.

624. In general (resistance to disease, rate of growth, number of generations per season) is most often sought in the improvement of cultivated plants.

625. A hospital has delivered a brachydactylous child to a couple in which the woman is normal and of wholly normal family, and the man is normal but with some brachydactylous relatives. Can the parents properly claim that the hospital made a mistake? (Yes, no.)

Chapter XXVII

626. (Superior, inferior) classes are contributing more than their proportionate share of the next generation.

627. Sterilization statutes have several times been declared unconstitutional because they were (punitive, class legislation, ineffective).

628. The business and professional classes have (smaller, larger) families now than a generation or two ago.

629. Between 1900 and 1930 length of life in the United States was (increased, diminished) (3, 8, 11, 19) years.

630. Methods already in use of encouraging larger families include (increased income at birth of each child, political preferment for large families, loans to young couples canceled at birth of children, low rentals for families with children).

Chapter XXVIII

631. The population of the United States (has, has not) reproduced rapidly enough since 1930 to maintain itself.

632. The birth rate in the United States has (risen, fallen) remarkably since 1921.

633. The United States now has more (young, middle-aged, old) people than a stabilized population has.

634. The movement of population in the United States has in general been from (city, farm) to (city, farm).

635. Intelligence tests have generally shown higher IQ among the (rural, urban) population than among the (rural, urban).

636. The reasons assigned by married couples for their voluntary childlessness have been mostly (eugenic, selfish) ones.

Chapter XXIX

637. Agglutinogen (A, B) predominates in Europe, (A, B) in southeastern Asia.

638. A European white person who is tall and fair probably also has (wavy, straight) hair, a (high, flat) nose, (blue, brown) eyes, and a (round, narrow) head, and lives in (Hungary, Sweden).

639. Armies are apt to differ from the civilian population more if the army is raised by (conscription, voluntary enlistment) than if it is raised by (conscription, voluntary enlistment).

640. Race is (sometimes confused, identical) with (nation, language, religious forms).

641. Broad skull is (a, an) (Nordic, Alpine, Mediterranean) character, and (high stature, swarthy complexion, blue eyes, stocky build, wavy hair) is Nordic.

642. There is a (Nordic, Celtic, Latin, Alpine, Caucasian, Slavic, French) race.

643. The earliest mulattoes in America replaced chiefly an equal number of (blacks, whites).

644. Among the distinguishing marks of races is (stature, locality, cephalic index, language, nasal index, texture of hair, religion).

645. Foreign-born recruits of the United States army in the World War received in general higher mental ratings if they were from countries of (northwestern, southeastern) Europe than if they were from (northwestern, southeastern) Europe.

646. Race hybrids are more apt to be social outcasts in (North America, South America) than in (North America, South America).

Chapter XXX

647. Up to about (1820, 1850, 1880, 1910) immigrants came to the United States mostly from (the same, different) parts of Europe (as, from) the early American settlers. (Underline only the latest appropriate date in the first parenthesis.)

648. Immigration into the United States from (Germany, Canada, Sweden, Mexico, Jugoslavia, Brazil, Hungary, Argentina, France, Nicaragua) is limited by federal law.

649. The foreign-born more than fulfill their quota in custodial institutions of the United States with respect to (epilepsy, tuberculosis, feeblemindedness, deafness, insanity, dependency).

650. Restriction of immigration into the United States began about (1848, 1882, 1917).

Appendix

651. The standard error of a mean is likely to be (larger, smaller) if the population ranges from 40 to 60 than if the population ranges from 45 to 55.

652. The standard deviation of a population ranging from 15 to 30 is likely to be (greater, smaller) than that of a population ranging from 20 to 30.

653. If the tallest Frenchman were 70 inches high, the shortest 54, and the tallest Swede 74 and the shortest one 62, the mean stature of Frenchmen would probably be (greater, smaller) than the mean stature of the Swedes, and the coefficient of variation of the French population (larger, smaller) than that of the Swedish population.

654. Uniformity being a desirable quality of shipped fruits, a crate of oranges having a standard deviation in weight amounting to 1.4 ounces would be worth (more, less) than another crate having the same mean weight and a standard deviation of 1 ounce.

655. A carload of apples having a mean weight per apple of 6.6 ± 0.09 ounces is worth (more, less) than a carload of the same variety and quality having a mean weight of 6.6 ± 0.22 ounces.

656. The standard error of a mean would be (larger, smaller) if the mean is determined from 1000 individuals ranging from 60 to 80 than if it is determined from 10,000 individuals ranging from 60 to 80.

657. The coefficient of correlation tells (why, whether) there is any connection between latitude on the earth and the number of fin rays in fishes.

658. A coefficient of correlation is of (more, less) value if its standard error is 0.008 than if its standard error is 0.017.

659. The mean stature of 100 men chosen at random from a population of a million is a (better, worse) indication of the stature of the whole million than is the mean of 500 men.

660. A calculated mean stature of 68.5 ± 0.41 inches, taken from a random selection of individuals, is a (better, worse) indication of the mean of the whole population than is a calculated mean of 68.4 ± 0.22.

LITERATURE CITED

ALLEN, C. E. Sex inheritance and sex determination in plants. *Am. Nat.* **66**: 97–107. 1932.

AMSCHLER, W. The oldest pedigree chart. *Jour. Hered.* **26**: 233–238. 1935.

ARASIMOVICH, V. V. Onasledovanii sakharistosti u bakhchevykh. (Inheritance of sugar content in cucurbits.) In Russian with English summary. *Trudy po Prikladnoi Botanike, Genetike i Selektsii* (*Bull. Appl. Bot., Genet. and Plant-Breed.*) Ser. 3, **5**: 5–31. 1934.

AVERY, A. G., and A. F. BLAKESLEE. Visible mutations from aged seeds. *Am. Nat.* **70**: 36–37. 1936.

BAKER, O. E. The outlook for rural youth. *U.S. Dept. Agr., Bur. Agr. Econ.* (Mimeographed, undated.)

BANKER, H. J. Distribution of school success in families. *Jour. Hered.* **22**: 373–380. 1931.

BARRONS, K. C. A defense of basic plant patents from the plant breeder's point of view. *Jour. Hered.* **27**: 475–478. 1936.

BATESON, W. Materials for the Study of Variation. New York. Macmillan. 1894.

BAUER, K. H. Zur Vererbungs- und Konstitutionspathologie der Hamophilie. *Deutsch. Zeit. f. Chirurgie* **176**: 109–184. 1922.

BAUR (1936). *See* Baur, Fischer, and Lenz (1936).

BAUR, E., E. FISCHER, and F. LENZ. Menschliche Erblehre. Munich. Lehmanns Verlag. 4th ed. 1936.

BEADLE, G. W., and B. EPHRUSSI. The differentiation of eye pigments in Drosophila as studied by transplantation. *Genetics* **21**: 225–247. 1936.

BELLAMY, A. W. Bionomic studies on certain teleosts (Poeciliinae). II. Color pattern inheritance and sex in *Platypoecilus maculatus* (Günth.). *Genetics* **13**: 226–232. 1928.

BENEDICT, F. G. The racial element in human metabolism. *Am. Jour. Phys. Anthr.* **16**: 463–473. 1932.

BERNSTEIN, F. Zusammenfassende Betrachtungen über die erblichen Blutstrukturen des Menschen. *Zeit. ind. Abst. Vererb.* **37**: 237–270. 1925.

BERRY, M. F. Twinning in stuttering families. *Human Biol.* **9**: 329–346. 1937.

BLAKESLEE, A. F., and T. N. SALMON. Genetics of sensory thresholds: individual taste reactions for different substances. *Proc. Nat. Acad. Sci.* **21**: 84–90. 1935.

BLUNN, C. T., and P. W. GREGORY. Linkage studies with curly$_2$ in the rat. *Jour. Hered.* **28**: 43–44. 1937.

BRIDGES, C. B. Salivary chromosome maps. *Jour. Hered.* **26**: 60–64. 1935.

———. The Bar "gene" a duplication. *Science* **83**: 210–211. 1936.

BRIEGER, F. Über die Bedeutung der Chromosomenverdoppelung für das Problem der Artentstehung. (Vorläufige Mitteil.) *Ber. deutsch. bot. Ges.* **48**: 95–98. 1930.

BRITTINGHAM, W. H. Artificial mutation Oenothera franciscana mut. tenuifolia, a gene mutation induced by radium treatment. *Am. Nat.* **70**: 41–42. 1936.

BRYNGELSON, B., and T. B. CLARK. Left-handedness and stuttering. *Jour. Hered.* **24**: 387–390. 1933.

BURCH, G. I. Heading for the last census? *Jour. Hered.* **28**: 203–212, 241–254. 1937.

CARTLEDGE, J. L., M. J. MURRAY, and A. F. BLAKESLEE. Effects of temperature and moisture on the increase in mutations from aged Datura pollen. *Genetics* **22**: 187–188. 1937.

CARTWRIGHT, W. B., and G. A. WIEBE. Inheritance of resistance to the Hessian fly in the wheat crosses Dawson × Poso and Dawson × Big Club. *Jour. Agr. Res.* **52**: 691–695. 1936.

CASPARI, E. Zur Analyse der Matroklinie der Vererbung der a-Serie der Augenfarbenmutationen bei der Mehlmotte Ephestia Kühniella Z. *Zeit. ind. Abst. Vererb.* **71**: 546–555. 1936.

CASTLE, W. E. The Y-chromosome type of sex-linked inheritance in man. *Science* **55**: 703–704. 1922.

———. Continuation of experimental studies of heredity in small mammals. *Year Book Carnegie Inst. Wash.* **33**: 266–268. 1934.

———. Continuation of experimental studies of heredity in small mammals. *Year Book Carnegie Inst. Wash.* **34**: 274–276. 1935.

———, W. H. GATES, S. C. READ, and L. W. LAW. Studies of a size cross in mice. II. *Genetics* **21**: 310–323. 1936.

CHAMBERLAIN, H. D. The inheritance of left-handedness. *Jour. Hered.* **19**: 557–559. 1928.

CHESLEY, P. Lethal action in the short-tailed mutation in the house mouse. *Proc. Soc. Exp. Biol. Med.* **29**: 437–438. 1932.

CLARK, J. A., and G. S. SMITH. Inheritance of stem-rust reaction in wheat. II. *Jour. Am. Soc. Agron.* **27**: 400–407. 1935.

CLAUSEN, R. E. Interspecific hybridization and the origin of species in Nicotiana. *Zeit. ind. Abst. Vererb. Supplem. Bd.* **1**: 547–553. 1928.

COCKAYNE, E. A. The origin of gynandromorphs in the Lepidoptera from binucleate ova. *Trans. Roy. Ent. Soc. London* **83**: 509–521. 1935.

COE, W. R. Sequence of functional sexual phases in Teredo. *Biol. Bull.* **71**: 122–132. 1936*a*.

———. Environment and sex in the oviparous oyster Ostrea virginica. *Biol. Bull.* **71**: 353–359. 1936*b*.

———. Conditions influencing change of sex in mollusks of the genus Crepidula. *Jour. Exp. Zool.* **77**: 401–424. 1938.

CONRAD, K. Erbanlage und Epilepsie. Untersuchungen an einer Serie von 253 Zwillingspaaren. *Zeit. Ges. Neurol. u. Psychiatr.* **153**: 271–326. 1935.

COOK, R. C. A year of German sterilization. *Jour. Hered.* **26**: 485–489. 1935.

CREIGHTON, H. B. White seedlings due to homozygosity of a deficiency in chromosome IX of Zea mays. *Genetics* **22**: 189–190. 1937.

DAHLBERG, A. A. Inherited congenital absence of six incisors, deciduous and permanent. *Jour. Dent. Res.* **16**: 59–62. 1937.

DAVENPORT, C. B. Sex-linkage in man. *Genetics* **15**: 401–444. 1930.

———, and A. G. LOVE. Army anthropology. (The Medical Department of the United States Army in the World War. Vol. 15.) Washington. Government Printing Office. 1921.

DAWSON, A. B. Effects of 2, 4-dinitrophenol on the early development of the frog, Rana pipiens. *Jour. Exp. Zool.* **78**: In press. 1938.

DEMEREC, M., and M. E. HOOVER. Three related X-chromosome deficiencies in Drosophila. *Jour. Hered.* **27**: 206–212. 1936.

———, and G. A. LEBEDEFF. The gene. *Ann. Rept. Dept. Genet. Year Book Carnegie Inst. Wash.* **34**: 40–43. 1935.

DOBZHANSKY, T., and J. SCHULTZ. The distribution of sex-factors in the X-chromosome of Drosophila melanogaster. *Jour. Genet.* **28**: 349–386. 1934.

———, and C. C. TAN. Studies on hybrid sterility III. A comparison of the gene arrangement in two species, Drosophila pseudoobscura and Drosophila miranda. *Zeit. ind. Abst. Vererb.* **72**: 88–114. 1936.

DOLL, E. A. The inheritance of social competence. *Jour. Hered.* **28**: 152–165. 1937.

DUNN, L. C. A secondary mutation from white to a darker allelomorph in Drosophila melanogaster. *Hereditas* **21**: 113–118. 1935.

———. Studies of multiple allelomorphic series in the house mouse. I. Description of agouti and albino series of allelomorphs. *Jour. Genet.* **33**: 443–453. 1936.

EATON, O. N. A summary of lethal characters in animals and man. *Jour. Hered.* **28**: 320–326. 1937.

EPHRUSSI, B. The behavior in vitro of tissues from lethal embryos. *Jour. Exp. Zool.* **70**: 197–204. 1935.

FISCHER (1936). See Baur, Fischer, and Lenz (1936).

FISHER, R. A. The Genetical Theory of Natural Selection. Oxford University Press. 1930.

———, and B. BALMUKAND. The estimation of linkage from the offspring of selfed heterozygotes. *Jour. Genet.* **20**: 79–92. 1928.

FORTUYN, A. B. D. Inheritance of harelip and cleft palate in man. *Genetica* **17**: 349–366. 1935.

FRETS, G. P. Family investigations on the heredity of eye color in man. *Proc. 6th Int. Congr. Genet.* **2**: 53–57. 1932.

———. The cephalic index. *Compt. Rend. Congr. intern. Sc. Anthr. Ethn.* p. 120. London. 1934.

FRYKHOLM, R. Familiäre Hornhautentartung, vererbt durch 6 Generationen. Klinisch nachgewiesen bei 13 Mitgliedern der 3 letzten. *Klin. Monatsbl. Augenheilk.* **94**: 76–81. 1935.

GLASER, S. Sweat glands in the Negro and the European. *Am. Jour. Phys. Anthr.* **18**: 371–376. 1934.

GOLDSCHMIDT, R. Untersuchungen über Intersexualität. VI. *Zeit. ind. Abst. Vererb.* **67**: 1–40. 1934.

———, and K. KATSUKI. Erblicher Gynandromorphismus und somatische Mosaikbildung bei Bombyx mori L. *Biol. Zentralb.* **47**: 45–54. 1927.

GOWEN, J. W. Anomalous human sex-linked inheritance of color blindness in relation to attached sex chromosomes. *Human Biol.* **5**: 130–134. 1933.

GREULICH, W. W. Sex ratio among human stillbirths. *Science* **74**: 53–54. 1931.

HALDANE, J. B. S. The Causes of Evolution. New York. Harpers. 1932.

———. A probable new sex-linked dominant in man. *Jour. Hered.* **28**: 58–60. 1937.

HATCH, M. H. The role of the individual in the history of science. *Biologist* **15**: 9–26. 1933.

HAYS, F. A. Studies on the inheritance of persistency. *Genetics* **21**: 519–524. 1936.

HOLLANDER, W. F. A three-factor autosomal linkage group in the pigeon. *Am. Nat.* **70**: 53. 1936.

HOLMES, F. O. Genes affecting response of Nicotiana tabacum hybrids to tobacco-mosaic virus. *Science* **85**: 104–105. 1937.

HOLMES, S. J. The effect of migration on the natural increase of the negro. A Decade of Progress in Eugenics. *Scientific Papers of the Third International Congress of Eugenics.* Pp. 119–123. Baltimore. Williams and Wilkins. 1934.

———. Human Genetics and Its Social Import. New York. McGraw-Hill. 1936.

HORNBACK, E. T., and C. F. DEGARIS. The inheritance of cataract in man, pedigree and interpretation. *Jour. Morph.* **54**: 347–363. 1933.

HURST, C. C. Genetics of the human mind. A Decade of Progress in Eugenics. *Scientific Papers of the Third International Congress of Eugenics.* Pp. 409–416. Baltimore. Williams and Wilkins. 1934.

ILTIS, H. Life of Mendel. (English transl.) London. Allen and Unwin. 1932.

IMAI, Y. Linkage studies in Pharbitis nil. *Zeit. ind. Abst. Vererb.* **66**: 219–235. 1933.

———. Recurrent auto- and exomutation of plastids resulting in tricolored variegation of Hordeum vulgare. *Genetics* **21**: 752–757. 1936.

———, and D. MORIWAKI. A probable case of cytoplasmic inheritance in man: a critique of Leber's disease. *Jour. Genetics* **33**: 163–167. 1936.

JAAP, R. G. Alleles of the mallard plumage pattern in ducks. *Genetics* **19**: 310–332. 1934.

JOLLOS, V. Studien zum Evolutionsproblem. II. Dauermodifikationen, "plasmatische Vererbung" und ihre Bedeutung für die Entstehung der Arten. *Biol. Zentralb.* **55**: 390–436. 1935.

KEELER, C. E., and R. SHORT. Hereditary absence of upper lateral incisors. *Jour. Hered.* **25**: 391–392. 1934.

KEMP, TAGE. Prostitution. An Investigation of Its Causes, Especially with Regard to Hereditary Factors. Copenhagen. Levin and Munksgaard; London. William Heinemann. 1936.

KOLB, L. The intelligence of immigrants as measured by tests. (*Proceedings and Addresses of the 56th Annual Session of the American Association for the Study of the Feeble-minded.*) *Jour. Psycho-Asthenics* **37**: 395–411. 1932.

KROEBER, A. L. Blood-group classification. *Am. Jour. Phys. Anthr.* **18**: 377–393. 1934.

LAMBERT, W. V. The evidence for inheritance of resistance to bacterial diseases in animals. *Quart. Rev. Biol.* **8**: 331–337. 1933.

LANDAUER, W., and L. C. DUNN. Studies on the creeper fowl. I. *Jour. Genet.* **23**: 397–413. 1930.

LANDSTEINER, K., and P. LEVINE. On the inheritance of agglutinogens of human blood demonstrable by immune agglutinins. *Jour. Exp. Med* **48**: 731–748. 1928.

LAUGHLIN, H. H. Immigration Control. In a Report of the Special Committee on Immigration and Alien Insane, Chamber of Commerce of the State of New York. 51 pp. New York. 1934.

LENZ (1936). See Baur, Fischer, and Lenz (1936).

LEVIT, S. G., and L. N. PESSIKOVA. The genetics of diabetes insipidus. *Jour. Hered.* **27**: 445–448. 1936.

LI, J. C., and Y. L. TSUI. The development of vestigial wings under high temperature in Drosophila melanogaster. *Genetics* **21**: 248–263. 1936.

LITTLE, C. C. The present status of our knowledge of heredity of cancer. *Jour. Am. Med. Assoc.* **106**: 2234–2235. 1936.

LORIMER, F., and F. OSBORN. Dynamics of Population. New York Macmillan. 1934.

LUSH, J. L. Progeny test and individual performance as indicators of an animal's breeding value. *Jour. Dairy Sci.* **18**: 1–19. 1935

MACARTHUR, J. W. Linkage groups in the tomato. *Jour. Genetics* **29**: 123–133. 1934.

MACKAYE, L. Mongolism in nonidentical twins. *Am. Jour. Dis. Child.* **52**: 141–143. 1936.

MACKLIN, M. T. The hereditary factor in human neoplasms. *Quart. Rev. Biol.* **7**: 255–281. 1932.

———. Inherited anomalies of metabolism. I. Diabetes mellitus. *Jour. Hered.* **24**: 349–356. 1933.

MADLENER, M. Eine Bluterfamilie. *Arch. Rass. Ges. Biol.* **20**: 390–394 1928.

MALZBERG, B. Mental disease among native and foreign-born whites in New York state. *Am. Jour. Psychiatry* **93**: 127–137. 1936.

MARSHAK, A. Growth differences in reciprocal hybrids and cytoplasmic influence on growth in mice. *Jour. Exp. Zool.* **72**: 497–510. 1936.

McCARTNEY, E. S. Acquired and transmitted characters in Greek lore of heredity. *Papers Mich. Acad. Sci. Arts Let.* **7**: 21–40. (1926) 1927.

McCLINTOCK, B. The production of maize plants mosaic for homozygous deficiencies: Simulation of the bm_1 phenotype through loss of the Bm_1 locus. *Genetics* **22**: 200. 1937.

McQUARRIE, M. D. Two pedigrees of hereditary blindness in man. *Jour. Genet.* **30**: 147–153. 1935.

MEDVEDEV, N. N. Poluchenie mutatsii u Drosophila melanogaster pod vliianiem kombinirovanogo vozdeĭstviia X-lucheĭ i soleĭ tiazhelykh metallov. (Artificial mutations under the combined influence of X-rays and salts of heavy metals in D. melanogaster.) *Trudy Instituta Genetiki* (*Bull. Inst. Genetics*) **10**: 211–222. 1935.

MENDEL, G. Versuche an Pflanzen-Hybriden. *Verh. Naturf. Ver. Brunn.* **4** (Abhandl.): 3–47 (1865) 1866. (Transl. in Bateson, Mendel's Principles of Heredity. Cambridge University Press. Pp. 335–379. 1913.)

MEYERS, M. T., L. L. HUBER, C. R. NEISWANDER, F. D. RICHEY, and G. H. STRINGFIELD. Experiments on breeding corn resistant to the European corn borer. *U. S. Dept. Agr. Tech. Bull.* 583. 1–29. 1937.

MOHR, O. L., and C. WRIEDT. A new type of hereditary brachyphalangy in man. *Carnegie Inst. Wash. Pub.* 295. 1919.

MULLER, H. J. A viable two-gene deficiency. *Jour. Hered.* **26**: 469–478. 1935.

MURPHY, H. C., T. R. STANTON, and F. A. COFFMAN. Hybrid selections of oats resistant to smuts and rusts. *Jour. Am. Soc. Agron.* **28**: 370–373. 1936.

MYERSON, A., J. A. AYER, T. J. PUTNAM, C. E. KEELER, and L. ALEXANDER. Eugenical Sterilization: A Reorientation of the Problem. New York. Macmillan. 1936.

PAINTER, T. S. A new method for the study of chromosome rearrangements and the plotting of chromosome maps. *Science* **78**: 585–586. 1933.

———. Salivary chromosomes and the attack on the gene. *Jour. Hered.* **25**: 464–476. 1934.

PARKER, M. C. Inheritance of resistance to the common mosaic virus in the bean. *Jour. Agr. Res.* **52**: 895–915. 1936.

PASCHKIS, K. Zur Erbbiologie der Blutkrankheiten. 2. Die Anaemia perniziosa. *Wiener Arch. inn. Med.* **29**: 125–150. 1936.

PATTERSON, J. T. The production of mosaics in Drosophila by X-rays. *Anat. Rec.* **47**: 394–395. 1930.

———, and M. L. SUCHE. Crossing over induced by X-rays in Drosophila males. *Genetics* **19**: 223–236. 1934.

PEARL, R. On the incidence of tuberculosis in the offspring of tuberculous parents. *Science* **82**: 539–540. 1935.

———. Multiplying man. *Yale Rev.* **25**: 584–602. 1936*a*.

———. Fertility and contraception in urban whites and Negroes. *Science* **83**: 503–506. 1936*b*.

PEARL, R., and S. A. GOULD. World population growth. *Human Biol.* **8**: 399–419. 1936.

PENROSE, E. F. Population Theories and Their Application with Special Reference to Japan. Food Research Institute, Stanford University. 1934.

PINCUS, G., and P. WHITE. On the inheritance of diabetes mellitus. III. The blood sugar values of relatives of diabetics. *Am. Jour. Med. Sci.* **188**: 782–790. 1934.

POLLOCK, H. M., B. MALZBERG, and R. G. FULLER. Is heredity a causative factor in the manic-depressive psychoses? A Decade of Progress in Eugenics. *Scientific Papers of Third International Congress of Eugenics.* Pp. 333–342. Baltimore. Williams and Wilkins. 1934.

POPENOE, P. The progress of eugenic sterilization. *Jour. Hered.* **25**: 19–26. 1934.

———. The German sterilization law. *Jour. Hered.* **25**: 257–260. 1934.

———. Motivation of childless marriages. *Jour. Hered.* **27**: 469–472. 1936.

POTTER, E. L. A hereditary ear malformation. *Jour. Hered.* **28**: 255–258. 1937.

POWELL, E. F., and D. D. WHITNEY. Ear lobe inheritance. *Jour. Hered.* **28**: 184–186. 1937.

RAFFEL, D. A genetic study of the reduction division in Paramecium aurelia. *Jour. Exp. Zool.* **66**: 89–123. 1933.

RAMIAH, K., and C. R. MUDALIAR. The inheritance of red pericarp color in rice (Oryza sativa). *Madras Agr. Jour.* **23**: 268–269. 1935.

REED, G. M. Inheritance of resistance to loose and covered smut in hybrids of Black Mesdag with Hull-less, Silvermine, and Early Champion oats. *Amer. Jour. Bot.* **21**: 278–291. 1934.

REED, S. C. Harelip in the house mouse. II. Mendelian units concerned with harelip and application of the data to the human harelip problem. *Genetics* **21**: 361–374. 1936.

RHOADES, M. M. Cytoplasmic inheritance of male sterility in Zea mays. *Science* **73**: 340–341. 1931.

RICHARDS, M. H., and R. M. BALYEAT. The inheritance of allergy with special reference to migraine. A Decade of Progress in Eugenics. *Scientific papers of the Third International Congress of Eugenics.* Pp. 314–321. Baltimore. Williams and Wilkins. 1934.

ROBERTS, E., and L. E. CARD. Inheritance of resistance to bacterial infection in animals. A genetic study of pullorum disease. *Univ. Ill. Agr. Exp. Sta. Bull.* 419. 1935.

ROBERTS, H. F. Plant Hybridization before Mendel. Princeton University Press. 1929.

ROBERTSON, D. W. Maternal inheritance in barley. *Genetics* **22**: 104–113. 1937.

ROSANOFF, A. J., and L. M. HANDY. Etiology of Mongolism with special reference to its occurrence in twins. *Am. Jour. Dis. Child.* **48**: 764–779. 1934.

SARTON, G. Introduction to the History of Science. Vol. 2. Parts 1 and 2. *Carnegie Institute Washington Publication* 376. Baltimore. Williams and Wilkins. 1931.

SCHEURLEN, W. Über die Vererbung der Pigmentdegeneration der Netzhaut und ihre Verbreitung in Württemberg und Hohenzollern. *Klin. Monatsbl. Augenheilk.* **94**: 761–792. 1935.

SCHMIDT, J. Racial investigations. IV. The genetic behaviour of a secondary sexual character. *Compt. rend. travaux laboratoire Carlsberg.* **14**: 1–12. 1920.

SHULL, A. F., and M. WHITTINGHILL. Crossovers in male Drosophila induced by heat. *Science* **80**: 103–104. 1934.

SJÖGREN, T. Vererbungsmedizinische Untersuchungen über Huntingtons Chorea in einer schwedischen Bauernpopulation. *Zeit. menschl. Vererb. u. Konstitutionsl.* **19**: 131–165. 1935.

SONNEBORN, T. M., and R. S. LYNCH. Hybridization and segregation in Paramecium aurelia. *Jour. Exp. Zool.* **67**: 1–72. 1934.

STADLER, L. J., and G. F. SPRAGUE. Genetic effects of ultraviolet radiation in maize. *Am. Nat.* **70**: 69. 1936.

STANTON, H. M. An experimental investigation of musical inheritance. Eugenics, Genetics and the Family. *Vol. 1 of Scientific Papers of the Second International Congress of Eugenics.* Pp. 239–242. Baltimore. Williams and Wilkins. 1923.

———. Stability of the Seashore measures of musical talent as shown by retests. A Decade of Progress in Eugenics. *Scientific Papers of the Third International Congress of Eugenics.* Pp. 54–66. Baltimore. Williams and Wilkins. 1934.

STREETER, G. L., E. A. PARK, and D. JACKSON. Hereditary vulnerability to dietary effects in the development of bone. *Science* **85**: 437. 1937.

STUBBE, H. Samenalter und Genmutabilität bei Antirrhinum majus L. (Nebst einigen Beobachtungen über den Zeitpunkt des Mutierens während der Entwicklung.) *Biol. Zentralb.* **55**: 209–215. 1935.

STURTEVANT, A. H. The effects of unequal crossing over at the Bar locus in Drosophila. *Genetics* **10**: 117–147. 1925.

THOMPSON, W. S., and P. K. WHELPTON. Population Trends in the United States. New York. McGraw-Hill. 1933.

VRIES, HUGO DE. Sur la mutabilité de l'*Oenothera Lamarckiana. Compt. rend. acad. sci. Paris.* **131**: 561–563. 1900.

———. The Mutation Theory. (Transl. by Farmer and Darbishire.) Chicago. Open Court Pub. Co. 1909.

WAALER, G. H. M. Über die Erblichkeitsverhältnisse der verschiedenen Arten von angeborener Rotgrünblindheit. *Zeit. ind. Abst. Vererb.* **45**: 279–333. 1927.

WARREN, D. C. Mapping the genes of the fowl. *Genetics* **23**: 174. 1938.

WASSINK, W. F. Cancer et hérédité. *Genetica* **17**: 103–144. 1935.

WEIR, P., and C. C. LITTLE. The incidence of uterine cancer in Jews and Gentiles. *Jour. Hered.* **25**: 277–280. 1934.

WIENER, A. S. Method of measuring linkage in human genetics; with special reference to blood groups. *Genetics* **17**: 335–350. 1932.

WINGARD, S. A. The development of rust resistant beans by hybridization. *Va. Agr. Exp. Sta. Bull.* **51**: 1–40. 1933.

WORZELLA, W. W. Inheritance of cold resistance in winter wheat, with preliminary studies on the technic of artificial freezing tests. *Jour. Agr. Res.* **50**: 625–635. 1935.

WRIEDT, C. Heredity in Live Stock. London. Macmillan. 1930.

WRIGHT, S. Evolution in Mendelian populations. *Genetics* **16**: 97–159. 1931.

———. Physiological and evolutionary theories of dominance. *Am. Nat.* **68**: 24–53. 1934.

YASUI, K. Studies on the maternal inheritance of plastid characters in Hosta japonica Ashers et Graebn. f. albomarginata Mak. and its derivatives. *Cytologia* **1**: 192–215. 1929.

ZIRKLE, C. The Beginnings of Plant Hybridization. University Pennsylvania Press. 1935.

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